THE SCIENCE OF SEEING

IDA MANN
AND
ANTOINETTE PIRIE

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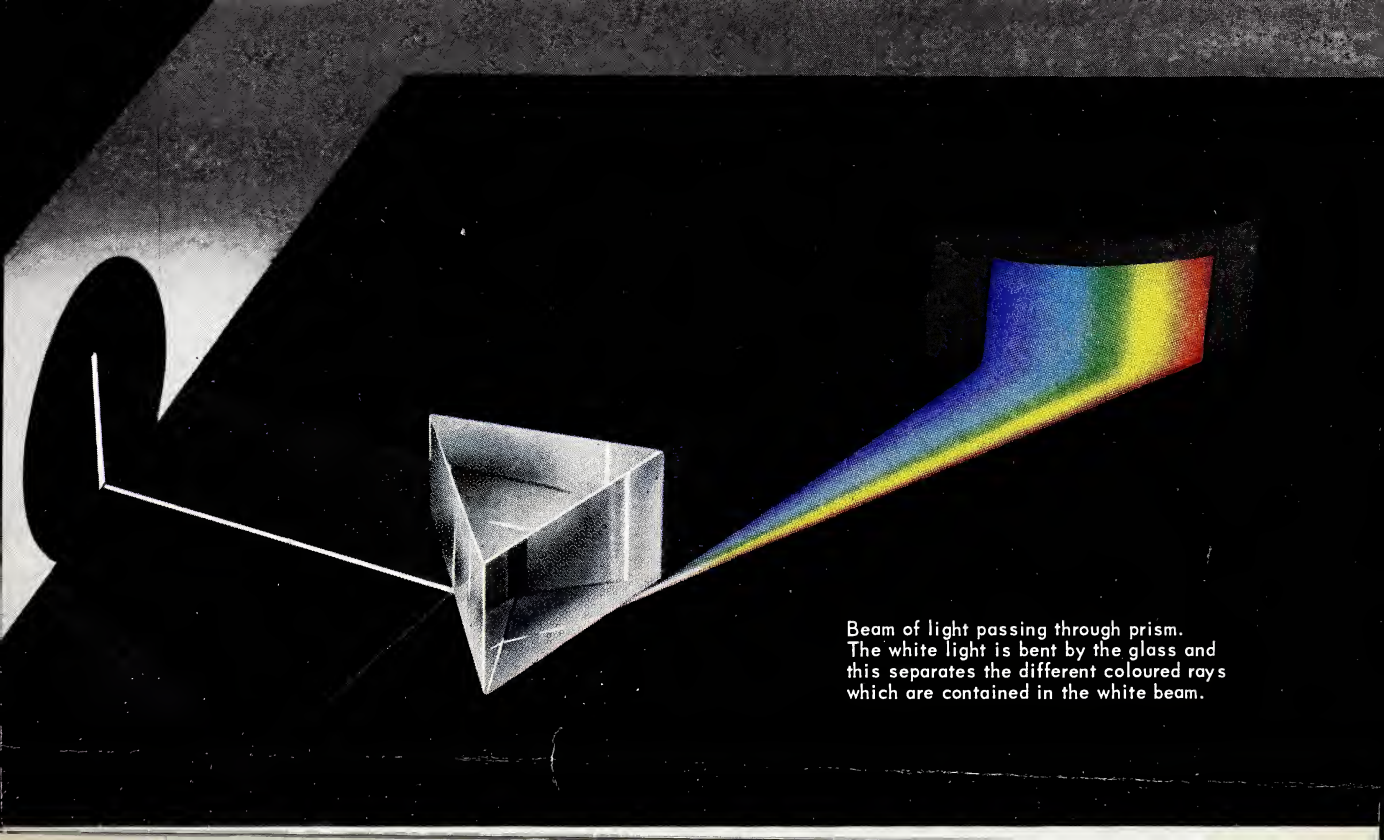

Beam of light passing through prism. The white light is bent by the glass and this separates the different coloured rays which are contained in the white beam.

THE SCIENCE OF SEEING

BY IDA MANN
AND ANTOINETTE PIRIE

'Why was the sight
To such a tender ball as th' eye confined
So obvious and so easy to be quenched
And not, as feeling, through all parts diffused
That she might look at will through every pore?'

MILTON: Samson Agonistes

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CHAPTER I

Grades and Varieties of Sight

To birds and men sight is the most important of the senses. But does the bird see the same world as the man? Almost certainly it does not, and if we could suddenly see with the eyes of a hawk or an owl we should have many adjustments of judgment to make before we could interpret the picture before us. We do not even know how the world looks to our neighbour, as anyone will realize who has gone picking strawberries with a colour-blind friend and has become involved in the hopeless attempt to explain the nature of the difference between red and green.

We know three facts certainly: that a form of energy (light) exists, and that it obeys definite physical laws, that all animals which 'see' have organs which are sensitive to this light, and that we ourselves have brains which, with more or less success, but in a quite mysterious way, sort out and interpret the meaning (to us) of the patterns formed by the light. The whole business is for most people at once so strange and so essential that it is not surprising that on the one hand a great many peculiar half-truths and imperfectly apprehended notions about how we see are current, and that on the other hand many fears, superstitions and anxieties arise in connection with sight and with the primitive terror of losing it. Too often we meet with loose conceptions about such things as the increase in the use of spectacles, the need or otherwise for 'care of the eyes', the best methods of 'preserving sight', and the possibility of decreasing the number of blind

people in the country. There are today approximately 76,500 of them in England and probably 5,000 in Australia, but we do not know this number accurately. Are they blind through ignorance of possible cures, lack of precautions, lack of medical treatment, or merely by an 'act of God?'. We try to look after them once they are blind, but we seldom try to understand how they have become blind and whether we might have prevented it. Indeed, to the average person, there is no clear distinction between true diseases which may destroy sight and other causes of 'weak eyes', such as errors of focusing. Neither is there any understanding of the sensations of partially sighted people, those people whose sight is not up to the usual standards but who are not blind. They form a class of the community for which very little is done, because they are suitable for some types of work and not for others, but few normally-sighted people understand exactly how much these people can see and so what jobs they are fit to tackle.

Our fears and ignorance are constantly being played upon by the suggestions of advertisements and our credulity fostered by stories of cures and systems, so that many people are prepared to believe the most peculiar things, such as that 'cataract is a skin that grows over the front of the eye', or that 'spectacles can ruin the eyes', or that 'if one eye is lost the other has to do twice the work', or that 'the surgeon took his eye out, scraped it and put it back', a statement often heard among hospital patients. There is, at the same time, a good deal of interest in zoology, and the question of how animals see is always fascinating. This book is an attempt to satisfy some of the average man's curiosity on these points.

Scientists are continually busy explaining things, but the first thing they always have to explain is that they will try to answer the question 'how?' but never, unless rashly lured into the forbidden ways of imaginative teleology*, the question 'why?' As it happens, the 'hows' of seeing and not seeing are mechanically simple, and a great deal is known about them. Scientists are also, in common with the rest of the world, fascinated by the idea of fitting everything into a pattern, to which end they try to survey the mulititudinous phenomena of reality as objectively and as widely as possible. They therefore prefer to consider sight as a sense found widely among all kinds of animals and differing in its quality and completeness from species to species, rather than as the perfected function of the human eye. When this is done, many of the disorders of the human eye will be seen to produce kinds of sight which would be normal in some other animals, a comforting thought when we realize how well the animal in question gets along. For example, all cats are colour-blind and all seals have severe astigmatism.

Since the pattern in which at the present time scientists mostly choose to arrange the animals is an evolutionary one, we shall find that if we are considering an animal function, namely sight, a similar arrangement is helpful.

The essential factor in sensitiveness to light (sight in its widest sense) is a chemical change. We are familiar with the fact that radiant energy of certain wave lengths effects a change in the chemical composition, and often in the appearance, of certain sub-

*Teleology—the doctrine that developments are due to the purpose that is served by them.

stances on which it falls. The common use to which this property is put is the photographic plate, on which an emulsion of silver salts is changed in proportion to the amount and kind (wave-length) of light falling on it, so that when the unchanged portions are washed off the plate a picture remains.

In living tissues under certain conditions a similar change occurs. Its primitive aim is to produce movement. This can easily be seen in the turning of leaves and flowers towards the sun, and in the movement of many small animals, which possess no true eyes, towards or away from light. This movement, phototropism, occurs even in the minute creatures, such as amoebae, in which the whole body consists of a single cell, and which therefore have no true sense organs. Some of them swim towards a light, others away from it, and the response is constant for the species. This cannot be considered as sight, but is the first step towards it, since it shows that light falling on a living cell can alter it and can make it do something. After all, to an inhabitant of Mars, watching us through a telescope, the only proof that we see would be through observation of our differing behaviour in light and in darkness.

It is obvious that when the whole surface of an animal (such as an amoeba) is sensitive to light, no visual discrimination is possible, other than that of the difference between light and darkness.

When, however, we begin to consider larger animals, composed of aggregates of many cells (multicellular organisms) we find that now, instead of the whole body being chemically affected by light, only certain specialized cells on the surface of the body

show this property. In the earthworm, for example, these cells are scattered rather indiscriminately over the skin, so that the earthworm, if it is wholly above ground, merely senses the presence of light, but, if only one end of it protrudes, may receive the impression of the direction of the light, since some of the sensitive cells will be stimulated and the others not. This indiscriminate scattering of light-sensitive cells over the skin is mechanically a very poor arrangement, and we soon find it superseded. In some animals (mostly marine worms and other water-living invertebrates) the light-sensitive cells are all aggregated together in a little patch, spread out flat on the surface. Such an animal may well sense, not only the presence of light, but also the direction from which it is coming, since one side of the patch may be more brightly lit than the other. Movement may also be appreciated, as shadows may pass across the organ (it is hardly an eye yet) and successive cells be exposed to the light. If the patch of cells is large enough, the shadow may form a definite pattern, and a sort of primitive sense of form be present. This is, of course, aroused only if a definite shadow shape falls on the eye. The shape of distant objects will not alter the evenness of illumination, since there is no mechanism for focusing the light.

The next stage is, therefore, the evolution of some means of focusing the rays of light from surrounding objects so that a picture or image of them is formed on the sense-organ. Such an image is formed on a photographic plate by the lens in a camera, or on the cinema screen, or on the screen of a camera obscura by the system of lenses in the roof of the room. In the eyes of animals various arrangements for produc-

ing such pictures are found (see chapters III, IV and V). Some of them produce only a blurred picture of outlines, others of greater complexity and perfection produce images as clear, accurate, and detailed as those of a micro-film camera.

Our animal now possesses appreciation of light, of movement, and of form. Such eyes are capable of still further evolution and improvement. So far we have assumed that light produces one kind of chemical reaction only (varying in intensity) in the group of sensitive cells on which it is focused by the lens system. But light is a form of radiant energy, and as such can be conceived of as a series of waves of varying lengths. Let our evolving animal develop the power of discrimination of different wave-lengths, and it has become sensitive to colour. At first probably only two colours will be seen, corresponding to the two ends of the band of wave-lengths to which it is sensitive. Gradually, with greater and greater discrimination, more and more intervening wave-lengths will be recognized as distinct, and more and more colours be appreciated. We see seven colours in the rainbow, but there is no reason to believe that no more are possible. It is even quite probable that some birds see more. After all, the number of different wave-lengths of which the rainbow is actually composed is infinite, so there is plenty of room for increased discrimination. Also, it happens that our light-sensitive cells are chemically attuned to a certain part only of the available radiant energy. There remains a great deal of which we are not conscious. A photographic plate can be produced which is sensitive to short wave-lengths beyond the violet end of the rainbow, or spectrum, and by a piece of chemical

jugglery an equally effective plate sensitive to infra-red rays can be made to take photographs under conditions of mist or fog, which render the objects entirely invisible to the eye.

To return to our evolving animal. It has now achieved sensitivity to light, movement, form, and colour. There still remains *perspective,* or the appreciation of the relative distance of objects from each other, and *meaning,* which depends on a comparison of objects seen with the memories of similar objects seen in the past. To take perspective first, we must consider two factors, namely, the focusing mechanism of the lens system and the nature of the field of vision. So far we have mentioned only that, in order to form a picture or image, a lens system is necessary to gather the rays of light and bring them to a focus. If we consider a camera we realize that rays of light coming from a very distant object will not be focused sharply on the plate together with rays from an object within a few inches of the camera, because in the one case they will be parallel, and in the other divergent, when they reach the camera lens. We have to adjust the camera (by moving the lens nearer to or farther from the plate) according to the distance of the object to be photographed. In the fully evolved eye this adjustment is enabled to take place by a variety of means in different animals. It is called the mechanism of accommodation. By the amount and kind of focusing required the animal may learn whether the object is near or far away.

Another factor, however, is envolved, and that is the field of vision. By the field of vision is meant the extent of the outside world which is visible at any time without moving the eye. The larger the field of

vision, the greater the number of objects in the picture at once, and the easier it is to determine their relative position. The things farther off will have smaller images than the near things, and also if the head (and eye) is moved, the farther-off things will seem to move relative to the near things, but in the opposite direction. This is known as parallactic movement. It is the sort of thing we notice when in a train going through a forest. The nearby trees rush wildly away behind us and the distant trees move forward with us in a sort of circular dance. By an artificial reproduction of this movement Walt Disney gets the illusion of depth into his animated cartoons, such as *Pinocchio* and *Bambi,* where we look down the long vistas of a mediaeval village or between the tree-trunks of primaeval forests. In certain animals the sense of perspective is still further enhanced by the overlapping and approximate superimposition of the fields of vision of the two eyes. Since each eye sees the scene from a slightly different angle, the two images are not exactly alike. The brain, however, perceives the two images as one (otherwise everybody with two eyes would see double all the time), but interprets the slight differences as being due (as they are) to varying distances of the objects from the eyes, and so gets an even greater sense of perspective, or depth. This faculty of depth perception by synthesis of two slightly dissimilar pictures is called stereoscopic vision, or stereopsis. It can be reproduced in photographs by taking two pictures of the same object from two points as far apart as the two eyes (three to four inches), and then looking at the pictures through an arrangement of lenses and prisms which present each picture to one eye only, so that the observer's brain must join

them. The figures in the photograph then appear solid. Stereoscopes when first invented were common objects of the Victorian drawing-room, and their effect was entirely beneficial. It is a pity that the good child is no longer rewarded by 'Peeps into Many Lands' on Sunday afternoons, since use of a stereoscope does much to improve judgment of distance.

Our evolving animal is rapidly approaching the ideal. He can see light, movement, shape, colour, and distance, and can vary his focus from the remotest visible star to a smut on the end of his nose. There remain but few further refinements. For one thing, he should be able to vary the amount of light entering the eye, stopping down the lens (as in a camera) to enhance definition and to avoid too much light. Also, he should be able to control all these functions of the eyes, the focus, the amount of light entering them, the angle between them, at will, as well as in response to the objects in the outside world which attract his attention. For this voluntary control as well as for the appreciation of the meaning of things seen he must have a good brain, and this question of the part played by the brain in seeing is so important that it demands a separate chapter. Indeed, the amount of use which an animal makes of its eyes and the amount of its control over them is very largely conditioned by the type of brain to which they are connected. This control varies with the species of animal, and with the individual. For example, birds have control over the size of the pupil, which men have not, and individual men vary very much in their ability to control the movements of their eyes and of their focusing mechanism. This is so, of course, with almost every movement of the body, some people being able to

twitch their ears or to move their toes separately, and others not. In some cases such movements can be acquired by practice.

So far, therefore, we must realize that there is more in sight than just seeing, and that it may be present in varying grades. These are: phototropism, sense of light, appreciation of direction and movement of light, of form, of colour, of perspective and sense of position and state of focus of the eyes, as well as of the meaning of the objects seen and the power to remember them and to take action on judgments formed because of them.

CHAPTER II

The Brain and its Relation to the Eye

It is possible to take an incubated hen's egg and, opening it very carefully, to detach that portion of the developing chick inside which will eventually grow into an eye. This tiny, rudimentary eye-bud can be kept growing in a glass container if it is at the right temperature and in a solution of the correct salts and foodstuffs. The little bud will grow and the various parts will form and the correct light-sensitive chemicals develop in it, till it is quite recognizable as a small eye, alive (since it is developing and growing) but not attached to any chicken. We can look at the little eye, but it cannot look at us, because, although the picture is formed in it by the lens and the right chemical changes occur, there is no brain attached to it to interpret the picture and no chicken to show appropriate movements in response to the dictates of the brain. So 'seeing', as we know it, implies the existence of some sort of brain.

In some of the primitive creatures we considered in the first chapter there is no true brain. Those whose whole body is a single cell may exhibit movement in response to the stimulus of light, but cannot vary this response. Here the cell which is sensitive to light is itself the cell which moves towards or away from the light; but as soon as the animal is multicellular, then we get a separation of the functions, certain cells being sensitive to light and certain other cells being capable of movement. It then becomes

necessary to connect the two in some way, so that the chemical change initiated in the eye by the action of light may be transferred to those cells of the animal which are capable of expansion and contraction and therefore of producing movement. To this end an intermediate kind of cell, which acts as a conductor, appears, and passes on the chemical change in the form of a minute electric current to the contractile cell.

Now we have an eye (sense organ), a nerve cell (conductor), and a muscle cell (motor), all linked up so that light falling on the eye produces movement. This simple chain of events is known as a reflex action, and is the basis of the majority of unreasoned and inevitable reactions of animals. As we climb up the evolutionary tree we find greater and greater elaboration of the nerve part of the pathway. More and more nerve cells become interpolated between the eye and the muscular system, and in this way finally an extensive and complicated network of nerve cells linked on the one hand with sense organs and on the other with muscles appears as the central nervous system, or brain. The brain then receives messages, in the form of minute electric discharges, from the eyes, and sorts them out, comparing them with stored memories of things previously seen and directing the currents of action to the appropriate muscles.

We can, therefore, recognize grades of seeing, beginning with the simple phototropism of the creature without nerve cells and passing up through the simple reflex action (the same stimulus always evoking the same response) to more and more complicated capacities for choice of action, as the animal stores more and

more visual memories and so reads more and more meaning into the picture formed in the eye. Quite complicated actions can take place in response to sight without any certain consciousness. The beautiful muscular movements of a seagull catching a piece of food thrown into the air are practically entirely reflex. The bird does not have to think them out; it sees the object thrown, the rest follows automatically. Birds, indeed, possess the keenest sight of all animals, but in the bird the part of the brain which we consider as the most likely to be the seat of consciousness is very small. The use we make of our eyes depends on the size and structure of the brain. A seagull has mechanically perfect eyes with which it can see the finest details of the smallest print, but no one would imagine that he could therefore teach a seagull to read. Conversely, the human race has developed the greatest consciousness and the biggest brain, and therefore the greatest possible number of alternative actions in response to any given stimulus, but it has not the best eyes. We can therefore learn to read, not because we have superlatively good eyes. but because we have a good enough brain linked to adequate eyes. Individuals, however, vary very much in the use they are able to make of the picture formed mechanically in their eyes. The famous aboriginal trackers of Australia have no keener sight than a normal European, but as the result of long practice they are able to attach meaning to small details which we ignore, though we see them equally well when they are pointed out to us. On the other hand, we can interpret and appreciate various forms of pictorial art which are meaningless for many primitive races. The power of aesthetic appreciation, which may be looked

on as the most complicated of our visual reactions, is largely the result of training, though it exists in a rudimentary form in all races, and also, curiously enough, in many kinds of birds.

But any day we can observe how different is the use made by different people of their eyes. There is a very true and unfortunately obsolete children's tale called *Eyes and No Eyes,* which brings out how two people seeing the same things can interpret them quite differently, and we have only to play the jewel game from *Kim* with a group of children and adults to realize how variable a quality is visual memory. Young babies see, but do not know the meaning of what they see, and occasionally from an injury to a certain part of the brain an adult may lose all visual memory and be reduced to the level of an infant. To such a person a teacup is a curiously-shaped blob of white or coloured light of unknown use, though if such a person takes the cup in his hands he will at once recognize it, since his tactile memory will help him out. A person born blind, whose sight is restored by operation in adult life, is in the same case, contrary to popular belief. The Victorian novelist's touching description of the delight of the heroine, previously blind from birth, when the bandages are removed by the clever surgeon after the heroic operation, are unfortunately for the poor doctor, purely imaginary. The patient is much more likely to feel slightly sick and to demand that the bandages be put back at once than to go into transports of joy. This is because the picture seen is utterly meaningless and therefore unpleasant, and a long period of training, during which the patient must link up touch and sight and work out the relations between them, as a baby does, is neces-

sary before she will be able to walk about with confidence.

We have all learned to interpret the pictures formed in our eyes by long experience in infancy. At birth a baby's eye is not fully grown, and the picture formed in it is not quite clear. The linking up of the two eyes is not complete either, so that at first the baby does not know how to direct its eyes towards the objects which attract its attention. One eye may move more than the other (which may make the mother suspicious that the baby has a squint or cast) and much practice is required before the baby can direct its hands and both its eyes towards the thing it wants. Even then it must learn the meaning of what it sees and grasps. The picture in the eye is not clear till the baby is six months old, because the light-sensitive layer has not finished growing, but after this has happened it should rapidly learn to co-ordinate its hands and eyes. Any squint present after this is abnormal and requires treatment. We have all forgotten our infancy, and so imagine that we always saw clearly and that the things seen always had meanings for us. We can sometimes, however, at the moment of waking, recapture our primitive amazement at the world for a few seconds, during which we see the usual things in the bedroom, but they look like something else, bizarre and meaningless. Our brains are not as awake as our eyes, and for a few seconds we cannot make any use of the picture we see.

On the other hand, a person with poor sight and a good brain may get on quite well, while another, stupider person with the same disability may be utterly helpless. This is because a good brain can supply by inference much of the detail which may be missing

to an imperfect eye. That we normally guess at much of what we think we see is apparent if we consider how we read a notice-board a long way off, or the name of a railway station as it flashes by. We know that the letters are those we are accustomed to, and if we just glimpse bits of them our brains can supply the missing parts. If the letters were in Hindi or Arabic script we should be quite unable to see the details of their pattern, because we should not know what to expect. Also, when we read rapidly we pay attention only to the general shape of the words and not to the component letters; indeed, it is quite possible to read if the top halves of the letters only are visible. Try covering the lower half of a line of print with a straight edge of paper; you can easily read it (if you cover the top half of the words it is not so easy).

It is the interrelation of mental capacity and physical condition of the eye which on the one hand makes it difficult to assess the exact point at which a person becomes legally blind (i.e. 'unfit to do work for which eyesight is essential') and on the other leads to a great deal of misapprehension about the amount to which the eyes can be used. It is not an uncommon experience to notice so-called 'eyestrain' after a long day of office work, study, or correcting examination papers, and yet to be able to spend the evening quite comfortably with a novel and an easy chair. The inference is obvious. It is not the eyes which are tired, but parts of the brain.

Emotional states, preoccupations, and fears are all capable of interfering profoundly with sight, the eyes themselves being normal. Many soldiers during the strain of war may suffer from a curious type of

blindness, in which the eyes are quite normal but the visual part of the brain pays no attention to the messages received from them. This was in most cases caused by some sudden shock during which the patient, so to say, forgot how to see, or in some cases was afraid to look. This condition usually recovers suddenly, sometimes in response to a second shock, and sometimes after treatment by suggestion, when it has the makings of a good miracle.

It can now be understood that, however perfect the eye is, its capacity for functioning will depend on the type of brain to which it is connected; and, conversely a first class brain can make very good use of an imperfect eye.

But sometimes a first-class brain is misled by a perfect eye, and an optical illusion results. We jump to a conclusion based on a preconceived idea rather than on the actual image presented to us. Thus the moon rising over distant hills may look enormous, though the size of the picture formed in the eye is exactly the same as that of the moon near the zenith. This is thought to be because the moon is judged against the hills, which are known by experience to

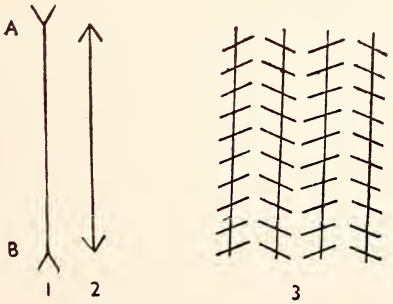


FIG. 1

be large. When seen near the zenith there is nothing, or only the branches of a relatively small tree, to which to relate its size. Similarly a man may look a giant when seen approaching through a mist. Look for example at the diagrams in Fig. 1 below. One's first thought is that the line AB is longer in 1 than in 2 and that in 3 the vertical lines are not parallel. A little measurement and a closer look will show the real state of affairs. We all realize that 'appearances are deceptive', and are not usually very surprised when this kind of thing happens.

We must now consider the structure of the eye in greater detail, so that we can get some idea of what we mean by a good eye and an imperfect eye.

CHAPTER III

The Construction of the Eye

Before we can form an adequate mental picture of the way in which an eye works, we must consider two inventions which, probably unknowingly, man has modelled on certain structures in the eye. One of them, the convex lens, is of great antiquity, and probably ante-dates any knowledge of the structure of the eye. The other, the photo-electric selenium cell, is a recent discovery, and its analogy to certain of the mechanisms of sight was well perceived from the beginning, as its popular name, the 'electric eye', shows.

To start with the convex lens. This is to-day a piece of glass ground and polished so that its surfaces are not parallel to each other, but are curved, each surface being a portion of a sphere. In other words, the lens is thicker in the middle than at the edges and is evenly curved. The use of such lenses to magnify objects and to set fire to objects by concentrating the light and heat of the sun on them was well known to the Chinese and to the Graeco-Roman world centries ago. They were then made of rock crystal and other natural transparent materials, and the memory of that period (which extended to comparatively modern times) still lingers in the term 'pebbles' used for spectacle lenses by old countrymen, and 'burning glass' used by children for hand magnifying lenses.

Light travels in straight lines in air until it meets a transparent surface of different density, e.g. a surface of water or of glass. It is then deflected or bent at this surface and continues on in a straight line in the medium (water or glass) at an angle to its original path. This phenomenon, called refraction, is well known, though we do not think much about it. No one is surprised that a straight stick looks bent when partly under water, or that a water flea looks as big as a rabbit when seen through a microscope. Both these appearances depend on this property which transparent materials possess of altering the direction of rays of light passing through them. Indeed much of the background of our modern life depends on the use of such arrangements (called lenses) to bend rays of light in such a way so to form pictures, which we then use for various purposes of pleasure, instruction and destruction. Cameras, microscopes, telescopes, cinemas, magic lanterns, reading spectacles, bomb-sights and periscopes are all possible because of this property of refraction and because, if the surface of the refracting substance is evenly curved and is convex, the parallel rays of light entering it will all approach each other and will, when they leave it again, form a small sharp picture of the object from which they came. The distance of this picture (or image) from the lens will depend on its curve. The more highly curved the lens is, the nearer the picture will be to it. This distance is called the focal length of the lens.

If you find all this rather muddling, go and get a small magnifying glass (there is always one somewhere in the house) or grandmother's reading spectacles, and try to catch the picture of a window or

an electric light formed by one lens on a sheet of white paper. There is only one position of the paper at which the picture is sharp, and that is where the paper is just at the focal length of that particular lens if the object is some distance away. If it is near the lens, but not within the focal length, the picture will be formed correspondingly further away. The best distance for experiment is five to six yards from the object to the lens.

You cannot see the picture (or image, as it is usually called) unless you catch it on a sheet of paper, but it is actually formed in the air and is there all the time, but invisible to us, since the rays of light forming it do not enter our eyes. If you find this idea of refraction, or bending of the rays of light by a piece of glass, very difficult to grasp, you can think of it in another way. Light travels fastest through a vacuum where there is nothing to hinder it at all, a little less fast through air, and still less fast through transparent substances of a density greater than air. Now, imagine your light rays as a regiment of soldiers marching over bare ground towards a wheat field, which is at an angle to their line of march. Obviously the soldiers cannot march quite so quickly through the wheat as over the open ground, and so their march will be slowed. The bare ground is air, the wheat field a piece of glass, and the files of marching soldiers are rays of light. As the advancing ranks meet the wheat field at an angle, the first file to meet it will be slowed up sooner than the next, and so on, so that they will pass through the cornfield with a slight deviation, which they will try to straighten up as they leave it. Fig. 2 shows how this works and Plate I shows the application of this to different shaped pieces

of glass, which can be built up to form a convex lens. Plate II shows a beam of light in a dark room focused by such a lens.

We have so far considered refraction as a property of such non-living materials as water, glass and crystal. There are, however, many living tissues or products of living tissues which are transparent, and so, given the appropriate shape, can act as lenses.

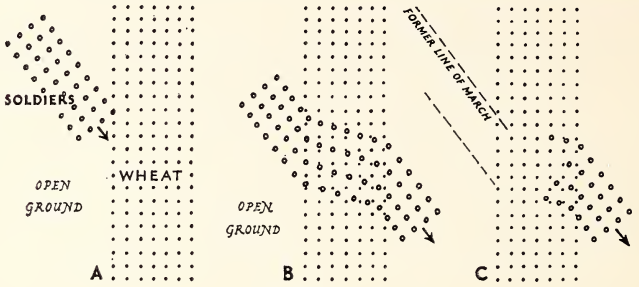


FIG. 2

Diagram illustrating the analogy of marching men being slowed down and therefore deviated by a field of wheat. *(See p. 27.)*

Horn in thin layers is almost transparent; the protoplasm of many of the primitive unicellular and multicellular animalculae is transparent, so that they are almost invisible in the water in which they live. The developing embryos of even the highest vertebrates, including ourselves, are in their early stages as clear as glass. Anyone who has opened an incubating hen's egg in the early stages knows that the chick is transparent and that its blood-stream and beating heart can be easily seen through the skin. So, transparency of living things is not a queer specialization, but rather a survival of an early and primitive state. Some

fishes even retain a good deal of transparency throughout life. There is one (called *Eviota personata)* which has transparent muscles and bones, even when adult. It is therefore possible to look on the transparent parts of the eye as the persistence of an almost universal embryonic state, the rest of the body having become secondarily opaque around them. Given the correct shape, certain kinds of living tissue can form lenses as efficient as those of glass.

The second device to consider is the self-generating photo-voltaic or photo-electric cell. This remarkable device which consists of a small plate of steel covered with a thin layer of a rare metal called selenium, and a further transparent electrical conductive coating, generates a tiny electric current when light falls upon its surface; within limits the amount of current generated is proportional to the intensity of light received. The self-generating photo-electric cell forms the basis of the photographic Exposure Meter wherein the cell is pointed at the scene to be photographed and a sensitive meter shows the magnitude of current developed by the cell. If the exposure meter is directed at a brilliantly-lit scene a high reading will be shown upon the meter, but if the scene is poorly lit only a small reading will be shown. The photographer can thus tell how to set his camera for a perfect exposure. Photo-electric cells of many types form the basis of modern methods of transmitting pictures by wireless or telegraph.

We therefore have a device which is capable of recording how much light it receives on a certain area. If instead of one large cell we imagine a flat mosaic of tiny photo-electric cells, each connected to its own recording mechanism, and on this we throw a picture

by means of a lens, then each cell responds to the intensity of light it receives and gives a reading upon its own individual recording unit. The cells in a brilliant part of the picture will generate more current than those in the shadows, and thus from these hundreds of small individual currents we can find out the degree of illumination at any part of the picture. If now we substitute a mosaic of light sensitive cells of the eye for selenium cells, and nerve cells connecting these to the brain for the electric wires which join the photo-electric cells to their individual receiving mechanisms, we have a mechanistic picture of the way the picture formed within the eye is transmitted to the brain.

The analogy is, however, lacking in one respect since the light falling on the photo-voltaic cell does not cause a chemical change in the normal sense, whereas the eye cells generate an electric current by producing a chemical reaction (remember the photographic plate again), which in turn starts the current in the nerves. In the photographic plate the chemical reaction started by the light cannot be undone, the silver salts are permanently changed, but in the eye the reaction is a reversible one, changing back again as soon as the light is withdrawn, so as to be ready for the next picture. This change to and fro is inevitable and requires no effort, neither does it wear out, however often it is made to occur. It happens just the same in the chick's eye growing by itself in the glass tube. Sometimes, if the light has been very intense, this change back is a little slow and we are bothered by a lag of the cells to get chemically adjusted again. This occurs over the whole scene when we go suddenly into a gloomy room from intense

sunshine and feel our eyes adjust themselves, or in local patches after we have looked at a bright light, or at the sun. Then we see a little dark or coloured 'after-image' of the light, dancing about in an annoying fashion and gradually fading. If the light has been excessively bright and hot, or has been watched too long, the chemical may fail to change back again owing to damage to the cells, and a permanent dark spot be left in the field of vision. Every time there has been an eclipse of the sun this has happened to a few foolish people, who have tried to watch it without properly smoked glass. It leaves a small, localized blind patch where some of the cells have been destroyed by the burning-glass action of the lens, and the result is called by the doctors 'eclipse blindness'. It is always therefore inadvisable to stare directly at the sun, though bright light is itself not harmful.

We have now a fair idea of the broad principles of seeing, and we can begin to think about the construction of various kinds of eyes. We shall see that they all have their parallel in various kinds of cameras, if they are capable of form sense at all. We need not bother much with those primitive eyes which see only direction of light and movement of shadows, except to notice two things. First, they are practically all dark brown in colour. This is due to pigment in the cells immediately beneath the light-sensitive cells, and is necessary to prevent undue loss and scatter of the light, which would blur the sensation. All cameras and optical instruments are made black inside for the same purpose. Secondly, the light-sensitive cells have microscopic hairs on them, called cilia, and these tiny hairs are along the border on which the light falls, so that it looks as if they might be the

site of the chemical change which starts the electric current. We do not really know much about this, but if we follow the evolution of these little hairs we shall find that in the higher animals they become the actual light-receivers which correspond with the free ends of the selenium studs in our photo-electric mosaic, so that they are obviously important from the beginning. It is also important for them to be very small and closely set together, rather like the pile of a very fine velvet. If the tiny receiving hairs, or the minute photo-electric selenium cells, are coarse and do not form a close mosaic, then the picture will be lacking in definition, as were the first telegraphed pictures in the newspapers. The closer and finer the mosaic, the more detail will be revealed, and the clearness of sight possible for any given animal or man ultimately depends on this close setting of the sensitive cells. In species of animals with poor sight the cells are large, in those with good sight they are very small. We ourselves have approximately a hundred and thirty-two million of them in each eye, and some birds have many more.

We must now return to the construction of cameras.

The simplest form of camera is the pin-hole camera, which some of us may have made as a toy. This is simply a closed black box with a bit of photographic film inside on one side, and on the side opposite to it a single minute hole. If a long exposure is given, an adequate photograph can be taken, since the small hole, by allowing only a tiny bundle of rays of light to enter from each part of each object outside, acts as a lens and forms almost as clear a picture. It has one advantage over a lens, namely, that within wide limits the distance of the camera from the object does

not matter, nor does the distance from the pin-hole to the film. The great disadvantage is the small amount of light entering and the dimness of the picture. A very practical application of the pin-hole principle can be tried by anyone who wears spectacles in order to see clearly (not merely 'rest glasses'). If you prick a very small hole in a card and hold it as close as possible to your eye in a good light, you will see almost as clearly as with your spectacles, only the light will be duller and the field of vision of course very small. This is a very useful trick for the over-fifty's, as one can read in a bright light at close range for a short time through a pin-hole without having to go and hunt for those elusive glasses.

A much more effective camera than the pin-hole one can be made by enlarging the pin-hole and putting a convex lens in it. Then the picture will be much brighter and the necessary exposure shorter. Most of us have possessed box cameras of this type, and very good pictures they took, on the whole. But they had the disadvantage that their focus could not be altered, so that objects very close to them could not be photographed clearly and objects very far away appeared extremely small.

The next improvement in a camera must obviously include some arrangement for altering the focus. One might do this by altering the curvature, and thus the power, of the lens, or by altering the distance of the lens from the plate. The latter is the usual method of focusing a camera, and leads to the concertina-like structure we are familiar with, which allows us to slide the lens backwards and forwards in response to the change in position of the object we wish to photograph. If we add to this some arrangement for stop-

ping down the lens in bright light (i.e. diminishing the amount of light entering the camera by means of the so-called iris diaphragm) and an adjustable shutter to control the time of the exposure, we have the modern efficient camera. This has fairly recently been improved still further by improving the quality of the lens, so that the picture (and so the whole apparatus) can be very much smaller without losing definition. The modern micro-film camera, the tiny pictures of which can be enlarged without loss of clearness, possesses all these refinements and is at the moment a fitting analogy for our purpose, since the actual size of the picture formed on the film is very near that of the picture formed in our own eyes.

But we are getting ahead too fast. What have cameras to do with eyes? Let us go back to our evolving animal and watch him trying out these various ways of making pictures, but in another chapter.

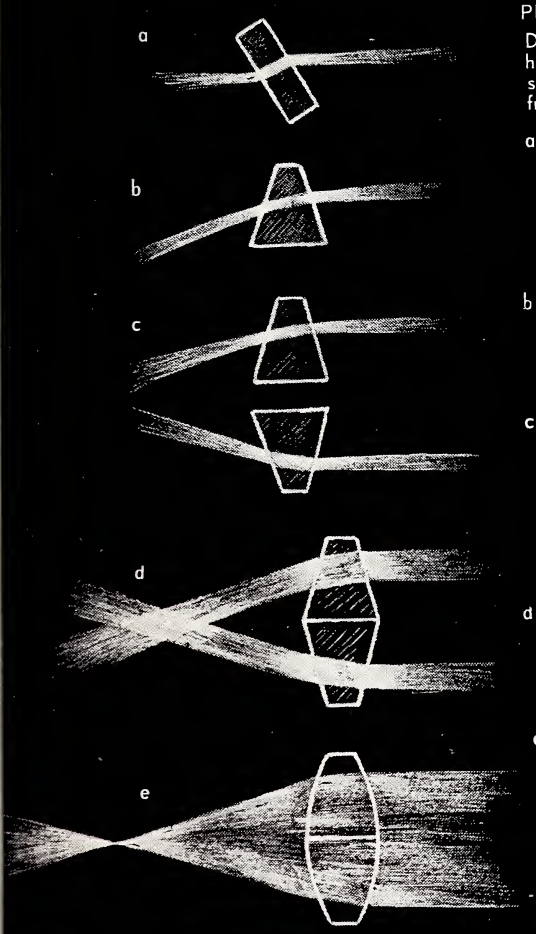


PLATE 1

Diagrams illustrating how a lens can be considered to be built up from two prisms–

a Light rays striking a parallel-sided piece of glass at an angle. The rays entering and leaving the glass are parallel to each other, but displaced.

b Shows displacement of light rays by prism. They are deflected towards the base of the prism.

c Effect of two prisms set base to base. Light rays entering parallel to each other are bent towards each other on passing through glass.

d The two prisms are set touching each other. The light rays overlap.

e The two prisms have their faces rounded so that light rays entering parallel to one another emerge convergent and form a picture at one point where they cross. Thus we have a convex lens.

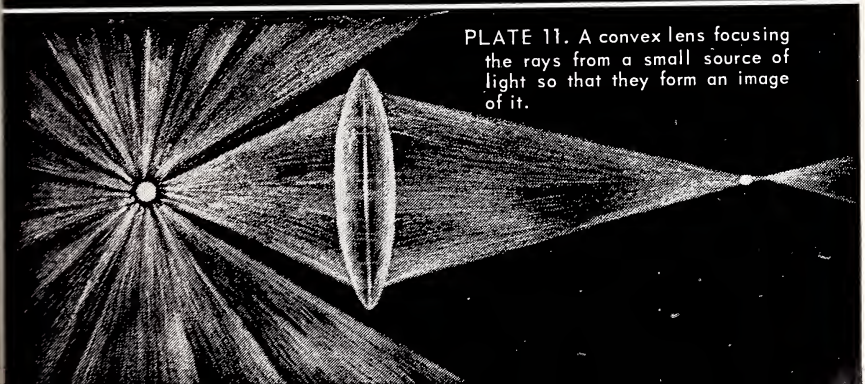


PLATE 11. A convex lens focusing the rays from a small source of light so that they form an image of it.

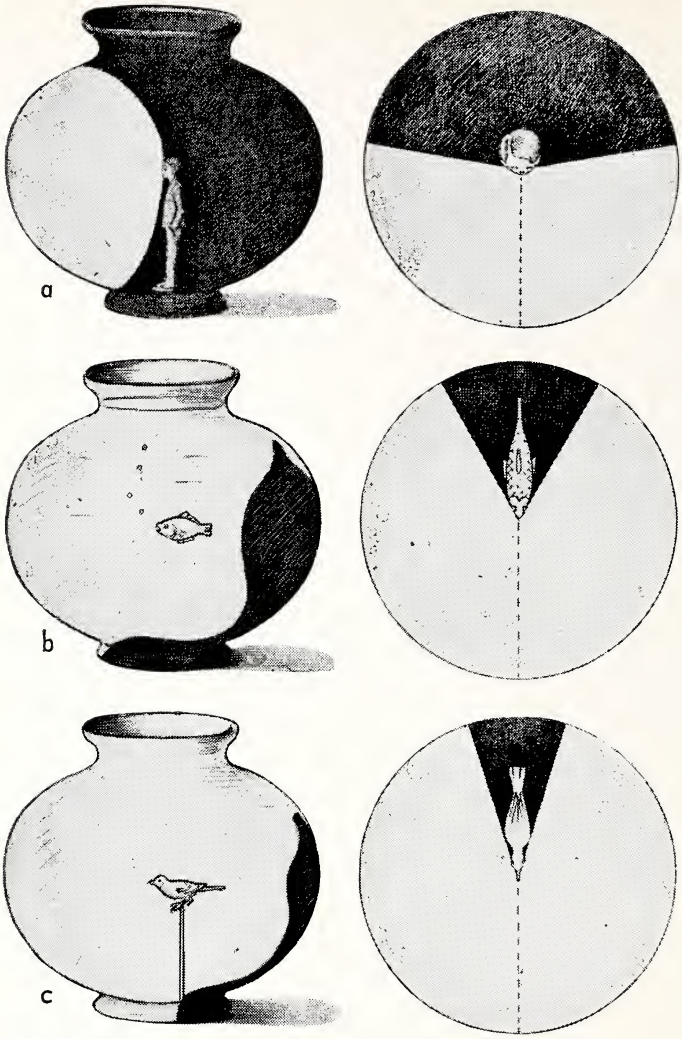


PLATE III, Diagrams illustrating the field of vision of a man, a fish and a bird. The creatures are represented inside glass bowls; the portion of the bowl which is darkened is invisible. The circular diagrams show the angle of the field of vision if the observer is above the bowls: *a*—man; *b*—fish; *c*—bird.

CHAPTER IV

Kinds of Eyes

The first advance from the primitive patch of light-sensitive cells on the surface of the animal occurs when, as in some shellfish (for example, the Nautilus or 'Portuguese man o' war', and, to a less extent, the more familiar limpet), the patch sinks below the surface, forming a little hollow sphere with a single tiny opening on the skin. The light-sensitive cells line this, and through the tiny opening the light rays enter, as through a pin-hole camera, and form a picture. These creatures have no true brain, but there are plenty of connecting nerve cells linking up the eye with the creature's muscles, so that it makes the appropriate actions when it 'sees' something. Only things which have a meaning for that kind of creature (food and enemies) will produce actions. One cannot expect a limpet to show any particular excitement if faced with a *Times* leader, or a Cézanne. Such creatures live in water, so it does not matter that the inside of the eye is open; the water just washes in and out. No sea-living creature has need of eyelids either, since the water washes the eye constantly in the same way as our tears and lid blinkings do.

The next step is seen in those creatures (such as squids and octopuses in the sea, and snails and slugs on the land) which close up the surface opening of their eye sphere entirely, but keep the skin over it transparent, so that the light can enter just the same. This patch of transparent skin thickens and gets a

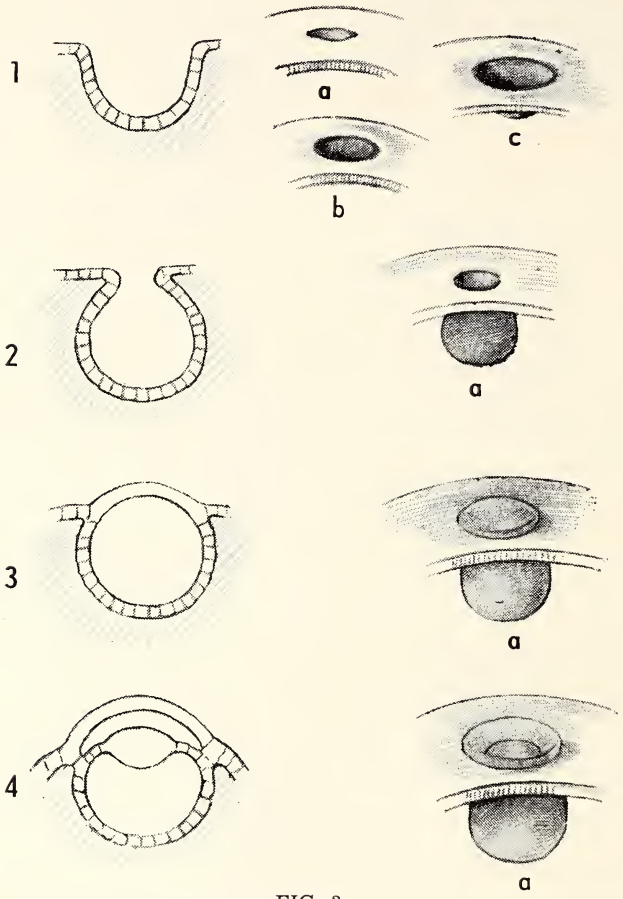


FIG. 3

Diagrams showing sections and models of various cuticular eyes.

1. Section through simple depression on the surface. 1a, 1b, 1c are models showing depressions of various depths.
2. Section of a cup-shaped eye partially closed and acting as a pinhole camera. 2a—model of this.
3. Section of cup eye which has closed in, the surface layer remaining transparent like a watch-glass. 3a—model of this, a vesicular eye.
4. Another type of vesicular eye in which a lens has formed beneath the transparent surface layer. 4a—model of this.

curve on it, and so begins to act as a lens and to focus a picture on the light-sensitive cells deeper in. Some of these creatures elaborate things still further by sinking the lens-like thickening of skin below the surface and forming a second transparent skin over the hole left by this. Some of the squids in this way produce a really efficient eye.

We can now begin to use names for these various parts of a primitive eye. The curved lining of light-sensitive cells inside the spherical eye is called the retina, the bit of transparent skin which sank below the surface to form the focusing mechanism must obviously be called the lens, and the transparent surface of the eye, which is also curved, and through which the light enters to reach the lens and be focused on the retina, is called the cornea. (This means the horny layer, because it is tough and dense and clear, like a thin slice of horn.) The bundle of fibres attached to the nerve cells and carrying the message back to the rest of the animal is the optic nerve. Now, all these parts of the eye are developed as specializations of skin which have sunk below the surface, and are therefore called cuticular eyes (or epithelial eyes, since scientists call the surface layer of any animal the epithelium) (Fig. 3). There are many varieties of these cuticular eyes, some of them very elaborate, and, as optical instruments, very efficient. None of them, however, is linked with a brain as we know it, since no animal without a backbone and skull has our kind of brain and spinal cord. All these animals are 'invertebrates', but get along quite well, because they do possess long chains of nerve cells and nerve fibres running in a series of relays all down their bodies, which enable them to move about and do the ap-

propriate things. Whether they have any consciousness of their actions we do not know; they do appear at times to have a sort of memory. There are hundreds of kinds of invertebrates, all divided up into different classes, and their shapes and habits and lives are of the most varied. To mention a few of them: these live in water—marine worms, lobsters and crabs, jelly-fish, barnacles, limpets, sponges, corals, starfish, sea urchins, squids, and very many others. These live on dry land—butterflies, bees, spiders, scorpions, ant-lions, etc., while many, like dragon-flies, live half their lives in water and half in air. They are a varied lot, and not all have eyes, and no two kinds have quite the same sort of eyes, though most of them are built on the same pattern. One large class, however, the insects, has broken quite away from tradition and developed a very curious kind of eye. In the *simple* eyes we have been considering there is one lens to form a picture on the whole retina, but in insects with *compound* eyes, every two or three retinal (light-sensitive) cells have their own tiny lens, so that not only do the light-sensitive cells form a mosaic, but the little lenses as well. How the creature copes with all the numerous tiny pictures being formed all over the eye we do not know, but it seems to manage it very well. We must notice these compound eyes in passing, but they have nothing to do with our own, or any other vertebrate eye.

Now when we come to the backboned (vertebrate) animals (e.g., fish, frogs, lizards, snakes, birds, rats, cats, tigers, sheep, monkeys, and ourselves, to mention a few at random) we find that the eye has got itself a bit mixed up with the developing brain, so that it does not start growing as a little surface patch that

sinks in, but as a little clump of brain cells that grows out and comes up under the surface of the skin of the head, which remains clear over it, and, as in the invertebrates, sinks in and makes a lens. So the vertebrate eye is in two parts, a cerebral (brain) part, which is the retina, and an epithelial (skin) part, which is the lens and cornea. This seeming complexity is produced because the whole brain of the verebrate is formed by the sinking in of a large area of skin at the head end, and the little eye-spots are on this brain area, so they get buried as well and, to be effective, have to bud out of the brain to find the surface and the light again. We need not worry much about this, nor about the consequent fact that in the vertebrates, owing to this early burying of the eye area, the retina is arranged inside out. In the invertebrates the little sensitive hairs, which we saw were essential to sight, stick out towards the light (i.e. the pile of the velvet is right side out), but in the vertebrates they point away from the light, towards the back of the eye. This does not matter at all, since the layers of nerve cells and fibres overlaying them are very thin and transparent, so that the picture is formed just as clearly, and, as the light-sensitive cells are on the whole smaller and better than those of the invertebrates, the final result is very good.

We must now see how the vertebrate eye works, i.e. what sort of camera it is and how good a mosaic of photo-electric cells it has, and what additions have been made to it for its protection and nourishment. We will start with fish. The fish has a retina in which the light-sensitive cells are fairly small, but not by any means the smallest known. It is obvious that the smaller these cells and the closer they are to each

other the greater the detail of the picture registered by them, so that even if certain of the fishes had good enough brains for the job it is doubtful whether they could read small print or thread a needle, because the picture would not show sufficient differentiation of detail. A whole group of fish, the Teleosteans (cod, salmon, herring, tunny, sword-fish, minnow, pike, to mention a few) have, however, achieved excellent eyes. The acuity of their vision in the central part of the field may be nearly as good as ours, and they can distinguish colours as well as we can. The more primitive orders of fish fall far short of this, however, and many which live in dark caverns and in the deep sea have lost their eyes altogether.

The fish of any class has, however, a good black layer of pigment outside the retina, and some fish have some shining cells here, which may be useful in amplifying very dim light by reflecting it from one layer to another. Outside the retina and the pigment is a layer of blood-vessels (called the choroid) whose function it is to supply oxygen and food to and remove waste products from the retina and the pigment. Outside this again is a tough fibrous layer (the sclera) which maintains the shape of the eye and protects the retina, which actually is a soft, thin, transparent sheet rather like a piece of wet tissue paper and no tougher. In the front part of the eye are the cornea and lens. Between the cornea and the lens is a neat, thin diaphragm with a hole in the middle through which the light passes into the eye. This is the iris, and is like the iris diaphragm on our camera, which is called after it.

The name iris and its German equivalent, 'Rainbow skin' *(Regenbogenhaut)*, refers, of course to the

variety of colourings seen in this part of the eye, which is most varied both in the individual animal or man and in the various species. It is this coloured part of the eye which attracts everyone's attention, and few people when looking at it notice the transparent cornea through which they see it. It has in most animals some little muscles in it which make the opening smaller when the light is bright (stopping down the camera lens) and larger when it is dull. The fish has not developed this arrangement, and the hole in its iris does not vary in size, nor is it perfectly round. The lens itself is almost round, like the glass ball in an old-fashioned ginger-beer bottle. You can easily find it in any fish's eye in the kitchen. If you wait till the fish is cooked you can still find it, but the heat will have turned it white and hard and opaque, so that it looks like a pearl and not a glass ball. Attached to the lens and behind the iris is another muscle which pulls the lens backwards and forwards (i.e. nearer to and farther from the retina) and so focuses pictures of objects at varing distances on to the retina, just as our concertina camera does. This pushing and pulling of the lens goes on inside the eye, not on the surface, as in a camera, because the eye has another transparent window, the cornea, which the camera has not. The cornea is a round, clear area in the sclera opposite the lens and the hole in the iris. It is rather like a watch-glass set into the opaque sclera. At the back of the sclera is another round hole, very small, through which the fibres of the optic nerve come out from the eye and go into the brain.

You can make a model of a vertebrate eye at this stage, and this will be helpful in visualizing how it works. A tennis-ball will do for the sclera. It is

round and tough and (possibly) white. Cut out a circular window on one side of it, into which you can later stick a watch-glass. Cut a small hole opposite this (but not exactly opposite the centre; lower than this and slightly to one side) and thread a bit of stout string through it for the optic nerve. The ball, except for the watch-glass area, must be lined with three layers; first a layer of thin red material to represent the choroid with all the blood in it, then a layer of thin black paper for the pigment, and then a layer of very thin velvet with the pile outside for the retina and its hair-like sensitive cells (or thin photographic film, if you want it to be more realistic, but this will not be easy to do and will be a waste of film). The end of the piece of string must be thoroughly frayed out and spread out on the surface of the retina to represent the minute nerve fibres picking up the picture from all the microscopic light-sensitive cells which are represented by the hairs of the pile of the velvet. As our eye is a vertebrate one the frayed-out string must be on the inside of the velvet; if it were an invertebrate one it would be underneath it. Now, cut a flat round piece of paper with a circular hole in the middle and fix a round glass marble in this hole. This is your iris and lens, and must be fitted into the front part of the ball just under, but not touching, the watch-glass which you are now going to fix into the opening in the front of the ball. If you have done this correctly and you now look through your watch-glass cornea into your model eye, you will find that you can see the paper iris and the front of the glass marble through the circular hole in the iris, but you cannot see through the marble into the inside of the ball. This is the same as in a camera, where you

cannot see beyond the lens, partly because of the black inside, and partly because the glass lens focuses any rays of light coming out so close to itself that you cannot catch them in your eye in ordinary daylight, because if you tried to, your head would get in the way and cut out the light. If you directed a thin beam of light in a dark room into the eye or camera and looked along the beam, you would catch some of the rays of light coming out and would see a little of the inside.

By this time it is obvious that the round hole in the iris in our model and in living eye is the pupil, and that the pupil is, so to speak, nothing at all, just a hole. Many people are very surprised by this, because the pupil of a human eye looks such a round, sharp, black object that it is difficult to believe that there is nothing there. This black hole has always been a bit of a mystery, and its curious name is an indication that it was early recognized as having something optically queer about it. Both the Latin word *pupilla* and also the Greek word *κόρη* for the same part of the eye mean 'little girl', and refer to the fact that if you look closely into your friend's eye you will see there a tiny sharp picture of yourself. This is really a reflection from the very smooth surface of the cornea, and is in front of the pupil, but may look as though it were in it. Under certain conditions the pupil may appear brightly coloured, and we shall deal with this later.

If you want to go into detail in the model, you must add two little circles of elastic, one round the edge of the pupil, to represent the muscle which makes it small in a bright light (and which fishes have not got), and the other at the base of the iris where it

joins the sclerotic, or outer coat of the eye. This is the 'ciliary muscle', and, by pulling on the lens to which it is attached by fine fibres, it changes the focus of the eye.

Now our model eye is almost complete, but we cannot complete it in fact (but only in imagination) without making a thorough mess of it. To complete it we should fill the narrow space between the paper iris and the watch-glass cornea with salt water and all the rest of the ball (behind the glass lens) with some clear jelly, such as the jelly part of frog spawn. A few more names are necessary. No self-respecting scientist would refer to the watery part as water, for it has a little salt dissolved in it and so is not pure water. It is therefore referred to as the 'aqueous' (which, after all, is only 'watery stuff' in Latin), and the jelly stuff behind the lens is called the 'vitreous', which means 'glassy stuff', but it is really much more like jelly than a bit of glass. The space filled by the aqueous is called the 'anterior chamber'.

Such an eye represents the basic arrangement of any vertebrate eye. The rays of light from the object to be seen pass through the cornea, where they bend inwards because of its curve, through the aqueous, through the lens (where they are bent still more), and through the vitreous, to make a chemico-electric kind of picture on the retina, which is sent along the optic nerve to the brain.

To return to our fish. Its eye is not nearly so beautifully spherical and well finished as our model. It is often rather flattened and its retina not very good, except in the cod and salmon group. The iris is, however, often very beautifully coloured, with all sorts of hues, gold, silver, red, green and mauve, which we

cannot boast. Also the fish can move its eyes slightly. To allow of this there are four (and sometimes six or more) narrow, straight little muscles attached like short bits of elastic at one end to the sclera and at the other to the bones of the orbit (the hollow in the skull in which the eye sits). By sending an electric nerve message from the brain along the nerve fibres to the muscles, first one and then another is caused to contract and the eye is turned from side to side. If we look at an ordinary fish, like a trout or cod (not a flat fish, like a sole or a plaice), we shall notice that the eyes are set on the sides of the head and not in front like ours. This means that the fish cannot look at any object with both eyes at once. It cannot therefore see stereoscopically. This is offset, however, by a very wide field of vision, each eye seeing almost a hemisphere of the surrounding water scenery, the hemispheres slightly overlapping in front and falling short of each other behind, where the fish's body gets in the way, so that there is only a little bit of the world immediately behind and below that is out of sight (Plate III). Also, since the fish has no eyelids, it cannot shut off visual impressions, any more than we can shut our ears even during sleep; this is, of course, one of the reasons for the controversy on whether fish do sleep or not.

The fish, therefore, has panoramic vision; it can see a large area, not particularly clearly and with not too sharp a perspective, and some fish can see colours. The world appears as a rather out-of-focus photograph in tones of grey, no one part being much more clearly seen than another, and the whole picture continuously present, though flagging of attention will have much the same effect as shutting the eyes has in us.

The fact that the fish lives in water and not in air affects the working of its eye camera in a curious way. Since the optical density of its cornea and of the water in which it swims is nearly the same, the cornea cannot be used to bend the rays of light, and the lens, which is denser than the cornea, must do it all. The light-rays just pass through the cornea as though it were not there, and therefore it does not matter what the curve of the cornea may be and whether it is rough or smooth. All that is necessary is that it be reasonably transparent. Hence we do not find among the fish the beautifully curved, highly polished surface of the eye which we see in land animals, but on the other hand we do find a very highly curved lens and rather curious focusing arrangements. These vary in different species. In some, the fish's eye at rest is focused for near objects, and when it wants to look in the distance (which of course it cannot often do if the water is at all muddy) it uses a little muscle which pulls the lens back towards the retina. This is the reverse of what most other animals do, whose eyes at rest make clear pictures of distant objects and who focus (or accommodate) when they want to see near things. These fishes, therefore, are rather like short-sighted people, who see near objects clearly but cannot see detail in the distance, but the fish is better off because it can focus the distance by moving its lens, while the short-sighted man cannot do anything except screw up his eyes to see more clearly by making a sort of pin-hole camera with his lids.

Other kinds of fish, however, are focused for distance when at rest and push their lenses forward to see things close to them. We can easily realize how living in water alters the optical properties of an eye

by noticing what happens when we dive and swim under water with our eyes open. If we have ordinary sight (good sight for distant and near objects) we see very badly in the water because we cannot use the cornea to bend the rays of light, but must rely only on our lens, which is not curved enough. But if we are short-sighted we see better under water, because doing away with the cornea bends the rays of light less, and in the short-sighted eye, as we shall see later, they are bent too much to start with.

There are very many queer modifications of eyes found in fish which live in very dim lights. If they live in completely dark caverns they often have no eyes at all, but if they live in the deep sea, where it is equally dark (no light penetrates through more than 500 yards of sea water), they often have enormous eyes with a very highly sensitive retina. This at first seems quite stupid until we remember that at these depths live many creatures which have developed phosphorescent organs. One can think of them as deep-sea glow-worms, to make this strange, black, silent world more credible. Here the fish must have an eye specially designed to catch the feeblest glimmer of pale phosphorescence. It does not require to see much detail, but it does want to see the least little bit of light; so here we come to one of the fundamental choices which an evolving animal must make in developing the kind of eyes it needs to suit its kind of life. Shall it sacrifice everything to sensitivity in order to be able to see in the 'almost dark'—no animal can see in absolute darkness, since there would be no light rays to set up the chemical and electric changes in its eyes—or shall it develop great discrimination for detail, for which good light is required, and sacri-

fice some night vision? This is the question of sensitivity *versus* acuity which interests all zoologists. Some animals have answered it in one way, some in the other; some, like us, have attempted a compromise. The deep-sea fish have gone all out for sensitivity and have lost colour and some form sense. They have enormously increased the number of light-sensitive cells. In some deep-sea fish there are 25 million of them to the square millimetre, while we have only 132 million in a whole eye. These fishes' cells are more sensitive to light than to shape. The surface fishes have, however, developed acuity for shape and colour at the expense of sensitivity. Each is suited to the life it leads, but we do not know which decided which—the kind of eye or the place the animal lives in.

CHAPTER V

More Kinds of Eyes

The tortoise incarnation followed the fish in the Vishnu story five thousand years ago, and the modern child with its illustrated encyclopaedia still pores over the mystery of the fish turning into the amphibian and crawling out on to the land in the latest version of the theory of evolution. Either way it seems to be accepted that something like this happened, and in the happening the creature had to modify its eyes. As soon as it got into the air, the cornea began to act as a refracting surface and so had to be smooth. The drying effect and the presence of dust made lids necessary for protection and as 'wind-screen wipers'. Also the cornea had to be kept moist, because if it got dry it lost its smooth surface and some of its transparency. Under these conditions land animals grew a set of glands* in various places in the lids which produce this necessary moisture and in the orbit around the eyes, and these secreted different kinds of fluids, some of them for wetting the eye, some of them slightly antiseptic to prevent injury by 'germs' (disease-caus-

*Glands may require explaining. The name comes from a word meaning an acorn, and is used in anatomy to mean all kinds of small or large structures which are rather lumpy and indeterminate in shape. Some glands function by producing a fluid (secretion) which runs from them through a channel, the duct, into some cavity, such as the glands which produce saliva and pass it into the mouth. Others, like the thyroid, have no duct, but put their secretion straight into the blood. A third kind (lymphatic glands) are part of the system of protection of the body and repel invading germs.

ing micro-organisms). Some of them nutritive (since the cornea has no blood-vessels and so cannot entirely look after itself), and some of them greasy to keep the margins of the eyelids smooth. So at once a move to another kind of life was accompanied by a number of new structures, and these in turn necessitated another, namely, a duct (tear duct) to carry off the excess of all these fluids into the nearest available damp space, the inside of the nose, which like the eye, had to develop a number of glands to keep it moist once the animal used its nose to breathe through, and not merely to smell with, as does the fish.

The amphibians seem to have decided that the dry land was worth while, and so finally took to it altogether and became reptiles. Some of these modified their eyes in various remarkable ways. Let us consider what some of them did about their lids. They all had scaly skins without hair or feathers, so none of them had any eye-lashes (which help to prevent bits of fluff and dust blowing into our eyes). Some of them (notably one kind of skink, like a slim lizard with short legs) took to burrowing just under the surface of sand. They shut their eyes, naturally, when doing this, but they also managed to develop a transparent scale in the skin of the lid through which they could see dimly even when their eyes were shut. This turned out to be a good idea for the snakes; they had no limbs at all, and so could not rub their eyes with their paws or protect them in any way, so they developed an even more transparent scale on a very thin eyelid indeed, and decided to keep their eyes always shut and to look through their transparent eyelids. They lost much clearness of sight in doing this, but gained much protection for their rather deli-

cate eye. Their eye scales get very scratched and dull, but that does not matter, for every time the skin is shed they shed the old celluloid-like eye scales, too, as a beautiful clear bright pair has formed underneath. You will surely have admired this arrangement if you have examined the shed skin of a snake.

The snakes, however, by making their eyelids so transparent, have reverted to the fish stage in being unable to open and close their eyes, but having better brains than the fish it is probably more difficult for them to ignore the messages which are continually arriving from their eyes. Some species have got over this by altering the shape of their pupils from round to a long narrow slit. This slit can be completely closed by the muscles of the iris, and so their eyes are as effectively shut by the iris as ours are by our lids.

There are numerous other modifications of this adaptive kind, involving the accessory things, such as the lids, the shape of the eye, the size of the cornea, the shape and size of the pupil, the colour of the iris, and the number, shape and size of the muscles which move the eye in the orbit. These things all vary from species to species and, in details such as the colour and pattern of the iris, between individual animals of the same species. There is therefore no limit to an account of the structure of animals' eyes if we go into detail. As our object is to understand the human eye and its functioning and why some people have good eyes and some bad and some go blind, we need not bother with animals except to show their variety and to get an idea of how we stand in relation to them from the point of view of seeing.

There are only two more classes of animals which

we must consider before we come to the mammals and man, namely lizards and birds: lizards because they are the common ancestors of men and birds and have eyes very like ours, and birds because they have succeeded in developing eyes which from a mechanical point of view are very much better than ours. Since the birds' brains have not developed along with their eyes and our brains have outstripped our eyes, the relative positions of men and birds in the world to-day are what they are. It is practically impossible for either to enter into the other's world of sensation, but man may more nearly understand the bird than *vice versa,* for a good brain is worth far more than a good eye in the struggle for existence, as a succession of blind celebrities from Milton to Sir Arthur Pearson demonstrate.

There are three important modifications of the eye itself which we must consider. The first concerns some improvements in the retina, the second is a change in the mechanics of focusing from the fishes' arrangement for pulling the lens backwards and forwards, and the third is in the position of the eyes, which come to the front of the head, so that the fields of vision overlap.

The lizards, and indeed a few of the fishes also, have elaborated the retina, which after all is the most important part of the whole machine. As we saw before, the clearness of the picture and the amount of detail in it will depend not only on the sharpness of the focusing by the lens, but on the smallness and closeness together of the cells forming the retina and on their sensitivity. In reptiles there is a patch in the retina (opposite the centre of the pupil) where the light-sensitive cells are slimmer and closer together

and so arranged as to be capable of registering a better picture here than elsewhere. A few of the fish also have the beginnings of this. As we have just such a little patch ourselves, almost identical in structure with that of the oldest living reptile (the Tuatera lizard of New Zealand—*Sphenodon*), it is quite easy to understand how this works.

Consider what happens as you read this page. You see quite clearly the word you are actually looking at, and also the one just before it and the one just after. The words which are further and further from the one you are actually reading are more and more blurred, though you can recognize the lines of print and the arrangement in paragraphs all over the page without directly looking at them all. You can also see a great deal more than this, though if you are interested in this book you will not be conscious of it until you take notice. You can see, though not sharply, your hands holding the book, the arms of the chair, the furniture of the room, the position of the fire and the light, if these are in front of you, and a host of things which fill your field of vision, but they get fainter and fainter towards the edges of it, that is to say, at right angles to the direction in which you are actually looking. This is because the light-sensitive cells are further apart towards the edges of your retina and very close together and more delicate in structure in the little central patch. This central patch is called a *macula,** which simply means a spot, and any animal which has one is at a great advantage because of the amount of detail which it can see and because its attention can be directed very intently towards one

*Its full name is *macula lutea* or yellow spot, as it has a yellowish tint in man. Its centre is called the *fovea centralis*, and is the point of most acute vision of all.

part of the field of vision. The Tuatera lizards and their relations have a macula in each eye, but as they have their eyes at the sides of the head and not in front (as, for example, have cats, owls, monkeys and ourselves), they have not got stereoscopic vision and do not see the same object with each macula, as we do. They therefore sacrifice some perception of depth for a larger field of vision with two (instead of one) areas of clear sight in it. Which they see is a matter of attention, and that is determined by the relative importance of the two objects to the lizard. It should be obvious that there is a difference between seeing and looking at a thing. One can look at a thing in the dark but not see it. Looking is turning the eye towards an object, seeing is perceiving it.

We all know by experience that if we want to see detail we look straight at a thing (i.e. put its picture on our macula), but must of us also know that if we want to see a very faint star on a dark night it looks brighter and we see it more easily if we look just a little off it and not directly at it, when, if it is faint enough, it may actually disappear. This looks as though our beautiful macula is letting us down; and so it is, because the very special cells in it will function only if the light is above a certain intensity. These cells, which see details in bright light, are slightly different in shape from the others, and are called 'cones' (because in some animals their terminal hairs are thick and conical), the others being called 'rods,' because they are more cylindrical. The study of their shapes is a very complicated microscopical business, and the names are not entirely justified, but their usage is so universal that we must adopt them. The cones, therefore, are bunched together very closely at the macula

and are scattered rather sparsely over the rest of the retina, while the rods are very numerous at the edges and are absent altogether in the macula. The cones function in bright lights and appreciate detail of form (and colour, in those animals which see colour), while the rods function best in dim lights, and only appreciate form rather vaguely, but are extremely quick to pick up movement. The dim star will disappear when its picture falls on a macula all made of cones, but will be seen as soon as it gets beyond the edge of the macula where there are some rods. The advertisement across the street will be deciphered by the macula, but the approaching motor-car will be picked up easily by the rods at the edge of the retina, which will see it move before they register its shape. We and the lizards orientate ourselves and move about among obstacles by means of our peripheral field (i.e. the whole area of the retina except the central or macular part) entirely. We examine the details of objects with our central field if the light is sufficient.

It is important to recognize these two different ways of seeing. The peripheral field is used entirely at night, since the central one is out of action. The central field is used entirely for reading, since the cones in the peripheral field are too sparsely set to register sufficient detail. We are not very conscious of these two kinds of sight (rod vision and cone vision, or peripheral and central, or scotopic [dark] and photopic [light] vision) until we lose one or the other. Suppose something goes wrong with the macula. The human being can be taken here as an example, since the retinal structure is so near that of the lizards. The person without a macula (both eyes are implied) will be in one sense blind, as he will not be able to

read even the largest print, to appreciate colour in the centre of the field, or to recognize people by their features, but he will not act like a blind man. He will be able to walk about freely, will not stumble over things, and on a dark night will be the equal of any of his friends. He will probably have to be educated at a school for the blind, as he cannot be taught by books, maps or pictures, but if he is, he will be found acting as guide to the other children. On the other hand, the person with a central field (and macula) and nothing else will be in an even more anomalous position. He will be able to read the finest print in good light, but will fall over the furniture in moving about a room and will behave as if blind when out of doors, and may be almost unable to find his way about. At night he will be totally blind. It is as if he sees through a long, narrow tube, and though the detail is sharp, there is never enough in the picture at once to be much help. Such a person may have to be certified as blind for purposes of employment as he cannot get about alone, though he is able to read.

We now come to the question of focusing, or accommodation, of the eyes for objects at varying distances. As soon as a macula has developed this becomes of importance, since unless the picture is sharply focused on the cones they will not be able to pick up the detail. There has been much controversy about this question of focusing and a great deal of popular misapprehension, due to the persistence in certain pseudo-scientific books on sight of theories based on experiments on fish and invertebrates only, whose methods of focusing are so different from those of lizards, birds and men that no inference can be drawn from them.

There are obviously many ways in which this focusing, or accommodation, for objects at varying distances can be arranged. We can discuss the theoretical possibilities quite briefly and see which animals have solved the problem in which of the many possible ways. It all seems rather overwhelming, but so is the whole animal world, if we stop to think about it, for we notice so many different ways of adapting to the circumstances in which the animal lives. Each kind of animal has hit on a different arrangement and nearly all the theoretical possibilities have been tried. Here are a few. First of all, the little hairs in the velvet pile of the retina have a certain length, and the picture will be appreciated if it is formed anywhere along that length (between the free and the attached end of the hair-like cell); therefore the longer the cell the greater depth of the picture in focus without the eye doing anything about it. In us the depth of focus we get by this means is from the distant horizon to within six yards of us (in normal eyes), but no nearer. In bats and octopuses (which probably have no other characteristics in common) this method is much elaborated, and they leave it at that; focusing is not an active business for them at all. There are two other ways in which this focusing can be arrived at without effort; one is by tilting the whole retinal surface relative to the lens and the front of the eye, so that the near objects focus on one part of the retina, the distant ones on another, and the animal has only to turn his eye to see either. This method is adopted among fish by the ray and among land animals by the horse; again we have a pair of utterly dissimilar creatures agreeing about the best way to see. The other method is by using the iris to stop down the

lens so much that a pin-hole camera effect is produced. Many animals do this. Thus the cat, by making a narrow slit pupil in daylight when definition of objects is important, and light not so important, and by opening the pupil to a circle at night to catch all the light there is, manages very well. Some animals which use this method aim at a slit, others at a small round hole, but the idea is the same. Sometimes the slit is upright, sometimes crossways. Go to the Zoo and see how many slit pupils you can find. It will be good hunting.

Now we come to the animals which do something active with their lenses when they focus. Some of the fishes, as we have seen, pull their lenses back to focus for distance, others push them forwards to focus for near things. Snakes and frogs also do this, and in all these creautres the lens is more or less of a round ball. Reptiles (except snakes), birds and mammals (we are mammals, rather primitive ones zoologically), however, have hit on another way still. They do not have spherical lenses, but bi-convex ones, like the lens of a powerful magnifying glass, and they alter the curves of the surfaces of their lens. This is possible because the material of which the lens is made is both soft and elastic, so that it can be pressed into another shape during accommodation, but will return to its original shape as soon as the pressure is released. The birds and lizards have very soft lenses which are squeezed by the iris and by the circular muscle at the base of the iris (the ciliary muscle), so that they become highly curved and bulge through the pupil when a near object is looked at. This gives them a tremendous range of accommodation; they can vary their gaze from the horizon many miles off to a grain of sand an inch or two from their eyes, and see each equally

clearly. This is very much better than anything we can do, for even an infant a year old (at which age the range of focusing power is greater than it ever is in later life) can only focus to a point between three and four inches from its eyes. In mammals the mechanism is somewhat different, since the lens is not squeezed in accommodation, but allowed to bulge by its own elasticity. We will say more about this presently.

It may have struck you that there is yet another way in which focusing might be done, and that is by altering the curve of the cornea and not of the lens. So far only invertebrates, and that rather underdeveloped fish, the lamprey, have adopted this method, though it is often ascribed by popular fancy to men, as is yet another way, namely by altering the whole back-to-front length of the eye by compressing the sclera by muscles from outside. This is quite untrue for any vertebrate. In all vertebrates the structure of the sclera is specially designed to prevent any change of shape at all. Some species, indeed have the sclera specially strengthened with bone and gristle to prevent even the slightest alteration; this method, therefore, does not concern us. The method of altering the curve of the surface of the lens is the one we are most interested in, and we must think about this in more detail.

When we considered the bending of rays of light by lenses we realized that the greater the curve of the surface of the lens, the more the rays of light were bent and the nearer to the lens the picture was formed (try a magnifying glass against a strong reading spectacle glass; the one usually forms a picture at a third of the distance that the other does). The con-

verse is true also. The nearer the object to be focused, the more curved must be the lens to produce a sharp picture at a given distance. Now the distance between the lens and the retina is fixed in any given eye, but the position of objects outside is always changing. The lens of fishes is spherical, like a marble, but in mammals and especially monkeys and man it is flattened, and has much the same curve as a good hand magnifying glass. It is not hard, but soft, and very elastic in texture. Its shape is due to its being firmly held by a tough ligament (suspensory ligament) all round its edge, which exercises a constant flattening effect on it. If the tension of this is relaxed or abolished the elastic lens tends to bulge and to become more spherical. So an arrangement is developed whereby the small ciliary muscle inside the eye (in a ring just behind the iris) can, by contracting, lessen the pull or tautness of this ligament and allow the lens to bulge. This happens whenever the mammal wants to look at a near object. This is the mechanism of accommodation in man, and it is a queer one, because it is, as it were, both a tension and a relaxation. The tension of the ligament is passive. It is a structural thing, and is there all the time the eye is at rest. It is roughly of such an amount that the curve of the lens it produces will focus objects six or seven yards or more away on the retina. If the object comes nearer, then the muscle contracts (which is an active tension) and loosens the pull of the ligament, allowing the lens to bulge (which is a passive elastic recoil), and so alter its focus. Work is therefore done by the ciliary muscle, but not by the lens or suspensory ligament. If, for any reason, the lens loses its elasticity it will not bulge even although the ciliary muscle

contracts properly and lessens the pull on it. This commonly happens in age, and used naturally to be put down to weakening of the muscle, until we came to understand that the change was a physio-chemical one and in the lens itself, which had become harder and less elastic.

Before we consider the mammals in detail, however, we must not ignore the birds, since they have far surpassed us in the beauty and efficiency of their eyes. In birds the range of accommodation is greater than in any other group, and they have developed two further improvements in their eyes. First, their eyes have moved towards the front of their heads, so that many birds look straight forward, as we do, and secondly, birds have elaborated the macula far beyond the lizard type, which is practically the same as the human.

This moving of the eyes to the front must be considered in conjunction with the improvement in the macular arrangements. It occurs in any group of animals when sight becomes the dominant sense and where accurate judgment of distance is essential. Thus we find it in all the hunting species, where the prey must be seen and pounced on, but not in the hunted, since they must have lateral eyes so as to get a wide field of vision in order that the hunter may not steal on them unawares. Hence the owl's eyes look forwards and the pigeon's sideways, the cat's forwards and the mouse's sideways, and so on.

In some birds—for example, swallows—the position of the eyes is such that the inner (nasal) halves of the fields of vision of both eyes overlap, and the bird sees objects straight in front with the two eyes stereoscopically, but can also see very far to the side with

each eye separately as well. In each of such eyes there are two areas of acute sight *(maculae)*. One of them in each eye is used when looking straight forward binocularly (with both eyes together) and stereoscopically; the other is used when looking at objects on either side of the binocular (overlapping) part of the field of vision. Hence the swallow can see with very great clearness not only the tiny gnat he is chasing in front of him, but also the whole landscape, two parts of which, one on each side, will also be seen in detail all at the same time. This sounds muddling for the bird, but actually is only part of his visual make-up, which is excellent. Some birds do better than this, and have their maculae spread out in long bands which run right across the retina, thus permitting a horizontal strip running right across the landscape to be all sharply in focus at the same time. It must be as if, standing in front of a long bookcase, we could read the titles of all the books from end to end without moving our eyes. Again, there is evidence from the arrangements and number of the nerve cells in the bird's retina that the acuity of sight is higher all over the field than it is in any other creature. Also, it is likely that the range of sensitivity for the infra-red or the ultra-violet is greater than ours. Nocturnal birds certainly can see much better in the dark than we can, and hawks in daylight undoubtedly pick up movements of small creatures in the grass from an incredible height. The old stories of sentinel vultures and the rapidity with which they notice each other's movements miles away bear out this reliance on sight. Man, indeed, has always recognized the superiority of the bird's sight, and has often used it to his own ends. Gordon Walls, in his book *The Vertebrate Eye*, says,

'it was the habit of the medieval falconer to carry a caged shrike on his saddle to keep track of the falcon. As long as the shrike acted fearful and excited the hawker knew that his proud tiercel was in sight, though not to him.' Small birds hunting for minute seeds and insects also require good sight, and it is indeed obvious both from their ways of life and from the structure of their eyes and brains that birds rely on sight more than any other sense, except the proprioceptive one.

Did I hear someone swallow hard over the end of the last sentence? This little-used word has been looming ahead for some time, and the idea behind it is ready to be tackled now. Shades of our grandmother's Guides to Knowledge! We thought we had five senses, and are always ignoring the sixth, yet without it we should not even be able to sit up in bed. Sight, hearing, taste, smell and touch are old friends, but we must not leave out the proprioceptive sense, which, unfortunately, has no popular name (though perhaps 'balance' comes near to expressing the idea). Its definition is the sense of passive position and movement of the body in space. If you wake in a pitch-black room and lie quite still, you are perfectly aware of the exact position of your whole body. You do not have to move your hand to find out where it is, or to turn on the light to discover whether you are lying with your knees drawn up or straight. If someone takes your hand and spreads your fingers out in the dark you can copy the movement easily and exactly with your other hand. This sense of the position of one's body is not the same as touch. It is allied to balance. It is the sense we make use of in riding a bicycle or skating or walking a tight-rope. But it is more than

this. It is the sense that enables us to make any movement at all, since it is quite impossible to move a limb if we do not know from what position we are about to move it. Obviously, one cannot start to walk unless one knows whether the legs are bent or straight to start with, as one would not know which of the movements of walking to make first. Some people who have partly lost this sense can learn to walk again by watching their legs and directing them with their eyes, but these people cannot balance or walk in the dark. This sense of position of the body comes from the muscles and joints themselves, and from a special balancing organ in the bone behind the ears. These send messages to the brain concerning the state of muscular stretch and the position of the head, etc. These messages are largely unconscious and we take them as a matter of course, but if we stop to think we can just feel all this going on immediately below the level of our attention. Some people are much better at perceiving this than others, and it can be improved by practice.

These proprioceptive messages from the muscles which move the eyes and from the focusing (ciliary) muscle are of importance to birds (and all the rest of us) in helping in rapid judgment of distance and quick changes of movement and directions. The swallow chasing his minute gnat two hundred feet up has need of every aid he can summon. Four maculae, stereoscopic vision, quick turnings of his eyes and head, and all the swiftness of his whole body will only just suffice to catch the gnat. If it were much easier the swallows would have eaten all the gnats long ago.

CHAPTER VI

Our Kind of Eye

The birds seem to have reached the top notch of sight, so where do the mammals (the other big offshoot from the lizards) come in? The mammals, the animals which suckle their young, are a varied lot. Just think of a shrew mouse and a whale, a kangaroo and a bull-moose, a giraffe and a marmoset, a yak and a man. All are mammals, so where do we begin? Why not with dogs and cats, the ones we see most of?

The slight inferiority of the mammalian eye shows from the beginning of its development. The little eye-bud is smaller in relation to the size of the head than in birds, and the eye when fully grown is much smaller relative to the size of the animal than in almost any other class. Consider the sizes of an owl and a man; yet of their two eyes, put side by side on a plate, the owl's would be the larger. Then, during the development of the mammalian eye before the animal is born there is a good deal that seems unnecessarily clumsy. First of all, the vitreous is at one time full of blood-vessels in the mammal (never in the bird) and these all shrivel up and disappear at birth and leave the vitreous jelly clear enough to see through. Often this clearing is incomplete and the vitreous remains full of little bits and tags of empty vessels floating loose. Secondly, the lens is never so elastic or so well developed as in the bird, and is covered before birth with a thin membrane of blood-vessels too. These also generally clear away, but in

many mammals (for example rats) some of them persist very often as a sort of cobweb across the pupil. All this clearing away is often not complete at birth, and in kittens and puppies the eyes are not finished enough to open for about ten days after birth. These tags of vessels left on the lens and in the vitreous finally do disappear, but the eye never develops as completely as a bird's. In puppies the retina, though it contains rods and cones, does not grow any little patch of specially good macula cones, and as the eyes (except in Pekingese and other artificially-bred flat-faced dogs) are still rather at the sides of the head, there is not specially acute vision or binocular stereoscopic sight. Anyone who has tried to show a dog a sitting rabbit down wind knows that it is a hopeless task unless the rabbit moves, when the dog sometimes picks up the movement. The dog will never chase the rabbit unless he smells it. Old dogs often go blind, but it is quite difficult to tell for certain that this is so, because the behaviour of a dog (which relies on its nose for everything) is nothing like so profoundly altered by blindness as is that of a man or a bird. A dog never shows any hesitation in greeting its owner whatever his appearance and attire, but a tame canary will often fail to recognize a well-known mistress if she greets it in a hat and gloves. Dogs therefore use their sight to avoid obstacles, but all detailed information about the world is got from their noses.

Cats, however, are better, as they show special adaptations to a night life and undoubtedly see better than we do in the dark. At once one thinks of glowing green eyes in inky jungles and of the lovely amber orbs, with their narrow black pupils, of our favourite puss, lazily stretched in the sun. If we want to observe

the reaction of the pupil to light we have only to watch a cat. The pupil is a narrow straight-up slit at midday and a huge wide-open circle at night, to let in all the light there is. But why do the cat's eyes shine green at night? The reason is that the cat's choroid (underneath the retina) instead of being a reddish network of little blood-vessels thinly covered by the pigment layer, has added to it a beautiful shining bright green layer called the tapetum (carpet). This is just under the equivalent of the pigment layer, which in animals with this kind of eye is not dark brown, but largely transparent. The use of this carpet layer is not entirely understood, but it is found in many nocturnal animals of all kinds (not only mammals) and possibly acts as a reflector in dim light, picking up what few light rays there are when these have passed through the retina and reflecting them back again on to the retina from behind, so that each light-ray hits its light-sensitive cell twice, once from in front and once when reflected back. This may double the available light stimulus and be very useful at night, but is a dazzling disadvantage in the day, and so the cat has to be able to narrow its pupil to the thinnest slit in sunshine, or the glare would be intolerable. The slit pupil also sharpens the picture, as we have already seen (Chapter V).

The green reflection from the back of the cat's eye is seen only in the dark when we are looking approximately along the beam of light which is entering the eye, as when we see the reflection of the car's headlights in a cat's eyes, as we drive towards it. There is, of course, no green glow if there is no entering light. The eyes are not self-luminous, and are quite invisible in complete darkness, contrary

to many people's belief. The tiger's eyes do not shine in the dark unless the intrepid hunter is carrying an electric torch or has lighted a fire, whatever William Blake and the Outpost of Empire story-tellers may say. This glowing or shining of the pupils is seen in all animals which have a tapetum (oxen, sheep, some dogs, all kinds of leopards and cats, and many others), and they are most of them nocturnal in habits. In most of them the reflected light looks green, or blue, and in many the tapetum is not present behind the whole retina, but only in a band, or an irregular patch. Even if there is no tapetum some light is reflected back out of the eye, and when one catches this reflected light in one's own eye the pupil can be seen to glow. If there is no tapetum this glow is red, from the blood in the vessels of the choroid, which shines through the pigment to a greater or less extent according to the amount of this.

Given the proper conditions (i.e. that the observer is looking directly along a narrow beam of light entering the eye in a fairly dark room) the glow of the pupil—called, in man, the 'red reflex' or reflection —can be seen in every mammal (and of course in other vertebrates, too), including man, but excluding the mole, which has only rudimentary eyes buried in fur and nearly closed at that. Very occasionally one catches sight of the red reflex if people are sitting round a camp fire at night and one looks through the firelight at the person opposite. It is most easily seen in certain rather uncommon shapes of eyes (eyes with large pupils and rather flat lenses), but it is quite easy to find it by experiment in everyone, man or animal. All you need is a small piece of looking-glass, say two inches square; scratch off a small

circular patch of the silvering from the back of the centre of the glass, just large enough to see through. Get your victim to sit facing you and to stare over your shoulder to the far end of the room, which should be dark. Put a small light (candle, electric torch, small electric light) just behind him so that his face is in shadow, but you can see the light. Now hold the piece of mirror by one corner and reflect the light on to his eye. When you can see the spot of light (like a heliograph beam) on his eye, put your own eye up to the back of the mirror and look through the hole you have scratched.* With a little patience you can easily catch the reflected red light coming back to the mirror from the choroid. It is a small, pale orange glow and rather surprising. If you have no luck at first with this experiment, try a cat or a dog and look for the brilliant green colour, which is very much easier to see than the rather uninteresting human reddish colour. The cat will probably not object, and you may be able to approach her closely enough with your little mirror to get a view of some reddish branching lines on the bright green background. They come out from the optic nerve which shows as a whitish disc if you have the luck to find it, and run all over the retina. Some (the arteries) bring blood into the eye, others (the veins) take it away again after it has helped to nourish the retina. These little arteries and veins are not present in the retinas of lizards and birds, which rely on the blood in their choroids for nourishing the retina, but most mammals, including man, have them, which is mechanically a pity because, as they lie on the surface of the retina, they interfere

* The ophthalmoscope used by doctors to examine the eye works on the same principle.

slightly with the clearness of the picture thrown on it by the lens.

Very few of the mammals (except monkeys and men) have a true macula, and in practically all of them the dominant sense is smell, not sight. Their eyes can be looked on as slightly degenerate, since their ancestors, the lizards, had far greater acuity of vision. Now man is a very primitive mammal in most respects, except for the size of his brain. He is soft, smooth, has no fur or hoofs or claws, and looks from an animal's point of view rather an unfinished embryonic kind of creature. He bears this out in the structure of his eye also. As mammalian eyes go it is a good one, but the points in which it is better are just those in which it most nearly approximates to the primitive lizard type, i.e. the possession of a macula and of a reasonably good power of accommodation. We must not, however, underestimate the wonderful capacities of the human eye. By quite remarkable compromises the eye, within the space of a cubic inch, possesses all the virtues of a camera automatically adjustable for distance, direction and variations of illumination; a combined low- and high-power microscope; and a photometer capable of appreciating differences of brightness from the lowest to those of a million times as great. It has no special arrangements, such as a tapetum, for seeing at night, nor is it specially large, and it shares with the rest of mammals the disadvantages of embryonic blood-vessels in the vitreous and in the pupil before birth and of blood-vessels on the retina throughout life. The reason why it is such a good instrument and so dominant in our lives is, of course, to be found in the beauty and complexity of our brains, which enable us to control it

so efficiently and to interpret its messages so fully; especially useful is our high power of synthesizing and fusing the pictures seen by our two eyes at once.

It is a popular fallacy that human beings look bigger to animals with big eyes, so that to a horse or a cow the world looks enormous and terrifying. This is quite untrue, as a little commonsense will show. The relative sizes of everything in the world look exactly the same to all the animals, whales, men, squirrels, bulls, and the rest. If they didn't, the animal would not be able to move about properly, as it would misjudge distances. The bull might think the grass was three feet high and start to eat at the wrong level, the squirrel might think that the branch he was leaping for was elsewhere than where it was and miss it, and the whale also might think the little fishing-boat was larger than itself and misjudge the moment to dive. No eyes 'magnify', since in all of them the picture formed is much smaller than reality, and the real size of the objects seen is determined by past experience, which links up touch and sight. We all know how hard it is to judge the height of a distant mountain or the size of an isolated shape seen at night. It is quite true that to a mouse a grass stem bears the same relation that a tree-trunk does to us, but the mouse sees his companions in the grass in exactly the same proportions as we see them. I do not see an elephant bigger than it is because it is bigger than I, so why should the mouse get mixed up over grass stems and trees?

Where the world does look different to animals is in its colour, its brightness and the amount of it visible at one time. Nearly all the nocturnal animals are colour-blind and see the world like a photograph.

Most birds have poor night vision and have gained colour vision with great discrimination of detail. Birds always get up and go to bed with the sun. They take no interest in things at night, unless they belong to the very special groups of night-birds, such as owls.

To the day-animals with cone retinas the world looks coloured, but not very bright; to night-animals (with rods only) it looks grey and very bright indeed by day and quite passably so at night. To those animals which, like us, have both rods and cones in their retinas the world has two aspects, a brightly coloured daylight one with a contral point of great detail, and an evening or moonlight one from which the colour has drained and the central point disappeared, but in which shadows and movements in the periphery are clearly seen. We resemble the lizards in this more than we do the majority of the mammals. Cats, dogs, rabbits, sheep, horses, and many others are colour-blind. So are bulls, despite the flaunting of the scarlet cloak of the torero and the widespread fear of crossing a field of cows when wearing a red coat. It is not the colour which annoys the bull, but the fluttering movement of the matador's cloak, which would act just as well if it were black or white or blue.

To animals with eyes at the sides of their heads a very wide field of vision is possible. We have a little over 180 degrees of field, or rather more than half the circle; a pigeon has 342 degrees, and a hare can see all round with an overlap between the two eyes, both back and front. A snipe can do this too, and so can a horse. It is, of course, very useful, as an approach from anywhere will be noticed at once without having to turn the head.

We can now consider the structure of the human eye in greater detail, specially emphasizing those parts of it which we can see and examine in each other, and about which there is so much unnecessary misconception and looseness of thinking.

First of all, let us look at the two eyes of a person. They are usually (not always) both the same colour, they both look straight forwards (unless the person has a squint, when one eye looks at you and the other somewhere else), and they can both move together, to the right, to the left, up and down, and finally they can both turn inwards, to look at an object held close to the nose (Plate IV). This turning in of the two eyes to observe a near object is called the movement of convergence. It is not the same as squinting, which is a disordered movement in which one eye is turned towards the thing looked at and the other is not (often popularly called a 'cast'). The eye at fault may be turned excessively towards or away from the other. Some people use the word 'squinting' to mean screwing up the eyes, but this is technically incorrect.

Both eyes are approximately on the same level, but very few people are mathematically symmetrical in this respect any more than they are in the shape of their faces. Both eyes move together. A favoured few can move one eye without the other, but this is a trick requiring practice; the power to diverge the eyes (i.e. turn the right eye to the right and the left eye to the left simultaneously) is very rare, though a few people can do it, at least wth one eye if not with both.

Above the eyes and lids are the eyebrows. Some people say they are mats to prevent sweat running into the eyes, others that they are the remains of the long sensory hairs (like the cat's whiskers) which

many animals have on their foreheads, and which can warn them of obstacles in the dark.

Now look at the eyelids. Along their edges are the lashes, the upper ones longer than the lower, and turned tidily outwards so that they do not tickle the eyeball. The upper lids drop in a rapid blink (to keep the cornea moist and clean) very frequently; in many people every two and a half seconds, in others less often. Shakespeare observed this and began one of his sonnets with the line, 'When most I wink then do mine eyes best see'. The edges of the lids are smooth and greasy, and if you look closely just behind the roots of the eyelashes you can see the openings of the ducts of the glands which make this grease. They look like a neat row of minute pin-pricks.

At the inner end (i.e. the end towards the nose) of each lid there is a larger hole. If you cannot find the ducts of the lid glands you will at least see this larger duct, especially the one on the lower lid. It is the opening of the tiny passage which carries off the excess tears and drains the eye into the nose. It is quite simply and sensibly called the 'tear duct'. The little passage from the upper opening joins that from the lower under the skin at the side of the nose, the passage widens, and then passes through a hole in the bone of the nose and opens near the floor of the nose. It is a common experience that when our eyes water, our noses require blowing. This is due to excess of tears running out of the drain. Sometimes the drain gets blocked up and the surgeon has to wash it out with a minute syringe, a proceeding which most people find very surprising, not having realized they had a hole through there at all.

In the inner corner of the eye is a little pink blob and a tiny fold of soft moist skin (mucous membrane). This is interesting, because it is all we have in the way of a 'third eyelid'. This third eyelid is another of the structures that the bird has elaborated. Some mammals (e.g. rabbits) have quite a good one, but if you have a parrot you will be able to see it well. It is a thin whitish membrane than can be moved over the eye from the inner side. Its movement looks very queer, like a rapid white sideways wink, and it is an elaborate and efficient windscreen wiper to keep the cornea clean. Ours cannot be moved; it is no use to us and almost invisible anyway. To those animals which have a good one the third lid can be useful. They may use it to protect the eye when going through sand or bushes. Bears, who have a very transparent one, use it as a kind of sun-glass to protect their eyes from glare off snow. Diving ducks have a very clever use for it. They have a thickened transparent inset portion, like a lens, in this third lid which they pull over the pupil when swimming under water. This counteracts the effect of the water on the focusing power of the eye and lets the duck see clearly through the water.

Now we come to the eye itself (Plates V and VII). We know now that it is a ball and that we only see the front part, the part of the sclera in which the cornea is set like the watch-glass in our tennis-ball model. The white of the eye is, of course, the sclera, covered by a thin, flexible skin with a few little blood-vessels in it. These vessels increase in visibility (because they dilate and fill up with blood) when we have a 'bloodshot eye' from any cause. The thin moist skin in which they are is called the 'conjunctiva' by anatomists and

inflammation of it is called 'conjunctivitis' by the doctors. This thin moist skin (or mucous membrane, as such stuff is called) covers the visible part of the sclera and then turns up and down on to the inside surface of the eyelids. You can see it by pulling the lower lid down, or if you are an adept at this useful trick, by turning the upper lid inside out. Get someone skilled in First Aid, or a nurse from an eye hospital, to show you how to do this, as it is useful to be able to get bits of grit out of the eye in this way. The gland which secretes the tears cannot be seen, as it lies above the eye under the bony ridge of the eyebrow.

The conjunctiva is not very sensitive. You can put your finger on it without much discomfort, but you cannot put your finger on your cornea without blinking at once, usually even before your finger gets there. The cornea, therefore, is very sensitive, and this is protective, because it is so important that it should be kept clean and bright, and we must know when the slightest speck of anything falls on it.

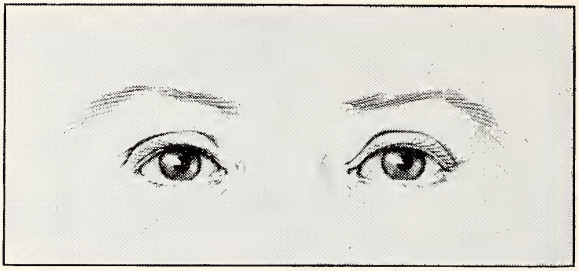
If you look at the cornea closely you will see that it is curved and transparent and that you are seeing the coloured iris and the dark pupil through it. If you look at the cornea from the side you can easily see that it is curved forward in front of the iris just as a watch-glass is in front of the face of the watch. In the watch also this curving becomes much more apparent when you look at the watch from the side; try this.

Now you come to the iris, which is fraught with interest and charm. First of all, it is coloured and has a pattern on it, and no two people have quite the same pattern or quite the same colour, except possibly identical twins. Also, the colour often appears to

change with the amount of light falling on the eye, just as the size of the pupil does. Flash an electric torch close to your eye in front of a mirror and you will see the pupil contract and dilate with the changing light.* The pupils will also contract when you accommodate and converge, in other words, when you look closely at something small. This is to increase the definition by stopping down the lens, just as you do with the iris diaphragm in your camera.

The colour of the iris is a kind of optical illusion, due to the presence of several layers of different colours superimposed. The back layer is dark brown, almost black, and is part of the black lining of the whole eye. There are some people and animals who have no colouring matter in their bodies at all. They have white hair, pink skins and pink irises with red pupils. They are called albinos, and are rather uncomfortable, as they have no protection against excessive light, and, if human, may wear tinted spectacles and carry sunshades. The albino animals (such as white rabbits with pink eyes) do not seem to mind so much, though albino monkeys often hold their hands over their eyes in the sun. The dark backing of the iris is, however, almost universal. The next layer is thin and white and rather stringy in appearance. It is made of little blood-vessels with thick walls so that the blood colour does not show. Blue-eyed people have these two layers only, the blue colour being really due to the white semi-transparent layer with the dark background showing through. There is no actual blue pigment in a blue eye. Most people have a third layer

* Both pupils contract though you only flash the light in one eye. Some few people have one pupil permanently larger than the other from various causes, known and unknown.



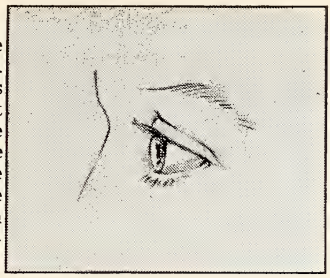
a. The two eyes of an individual looking straight forward at an object in the distance.

b. The two eyes converging and looking at a near object.

c. A squint of the right eye. The individual is looking at an object in the distance with the left eye and turning the right eye inwards towards the nose. This is a right convergent squint.

Plate V

The parts of the human eye which can be easily seen. The illustration below shows the right eye from the front with the names of the visible parts. The diagram on the right above shows the left eye from the side. Note how the cornea is bowed forward in front of the iris like a watch-glass in a watch.



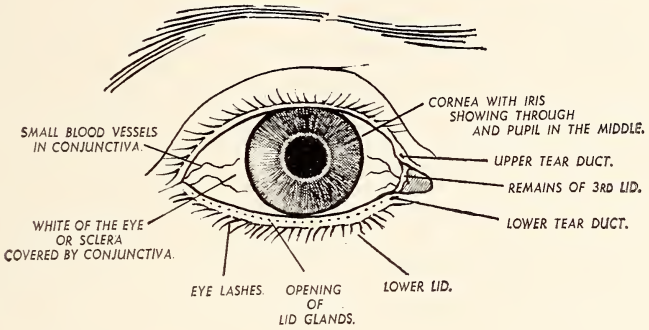
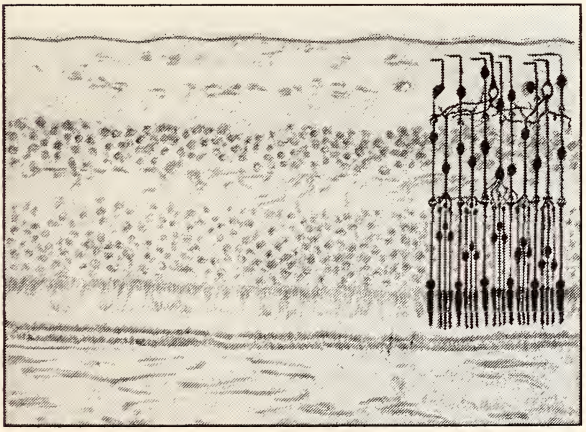

SMALL BLOOD VESSELS IN CONJUNCTIVA.
CORNEA WITH IRIS SHOWING THROUGH AND PUPIL IN THE MIDDLE.
UPPER TEAR DUCT.
REMAINS OF 3RD LID.
LOWER TEAR DUCT.
WHITE OF THE EYE OR SCLERA COVERED BY CONJUNCTIVA.
EYE LASHES.
OPENING OF LID GLANDS.
LOWER LID.

Plate VI. A microscopic section of the retina, with the "wiring diagram" showing the connections of the cells, superimposed on one end of it.



in front, more or less fully developed, of golden brown pigment. If this is present in a thin layer or in spots only we get the varieties of hazel, grey and speckled eyes, and if it is present in a thick sheet all over we get the dark brown or almost black eye. The speckled and partially pigmented and light brown types are the commonest in England, but no two irises have quite the same pattern. This has long been known, and Bertillon suggested using it as a way of identifying criminals. It has also given rise to endless fantasies and superstitions. People have given a magical interpretation to the little pigment spots, reading them into letters and words (rather like fortune telling with tea leaves) which have foretold events or accused criminals or forecast the individual's future. The name of Napoleon was frequently deciphered by the credulous of the irises of children born at the time of Waterloo, and even today a queer cult survives (mostly in Central Europe) called 'Iridodiagnosis', which attributes to each minute area of the iris an affinity with a different part of the body. The high priests of this mumbo-jumbo therefore claim that by studying the pattern of the iris they can tell what is the matter with the patient. This could not possibly be true, since the iris pattern is complete and permanent (save for certain rare diseases) at a year old.

This surface layer of pigment is not present at birth in Europeans, so all their babies have blue eyes. This blue is usually a lovely rich violet shade, due to some of the blood colour showing through the vessels, whose walls are thinner than they will be later. The proud mother is always disappointed to see this violet colour fade in a few months and change to light blue, grey, hazel, or brown.

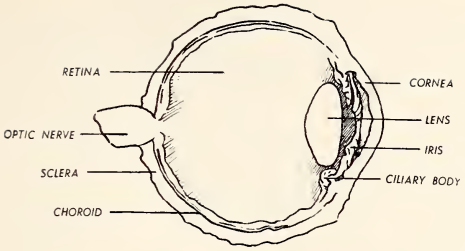
If we look at the pupil in ordinary daylight it looks black, but if light (from an electric torch, for example) is focused on it we can sometimes see a greyish tint on it and catch a fleeting glimpse of the front surface of the lens. The lens is very like that of the lower animals, but is not so elastic as that of the birds and lizards, and so has not so great a range of focus. The lens is held in the usual way by its suspensory ligament, which is relaxed and tensed by the ciliary muscle contracting and relaxing, as in most of the other mammals. The ciliary muscle is very strong and active, and contracts and relaxes with great rapidity more or less automatically for sixteen hours a day without fatigue in health. We are not usually conscious of its workings, but with practice one can acquire some control over it. In normal people, however, it acts quite automatically in response to the desire to look at any given object, and is as untireable as the heart, or the breathing muscles.

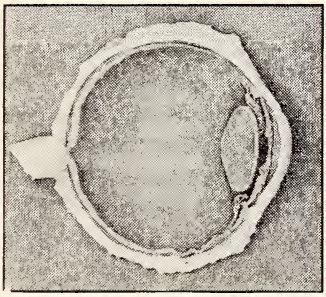
Behind the lens is the vitreous. In it there are little specks and strings of cells which are left behind from the blood-vessels which were present in it before birth. We all have these little remnants and can see them if we look at a clear sky or at the grey evenness of a fog. Most people, however, ignore them completely, realizing subconsciously that they are part of themselves and so of no import, like the beating of our hearts. Sometimes one notices them suddenly after years and thinks something has gone wrong because there are little dots and strings of clear beads and threads of fluff floating about in the sky or on the page of a book. Some people, however, have been familiar with them all their lives and do not worry. As we get older the vitreous jelly gets slightly more

liquid and they may change their position, float suddenly into view and so be noticed for the first time. The best thing to do with them is to ignore them and they soon cease to worry one. They are called 'floaters' or *muscae volitantes* (flying flies), and have been recognized for centuries as part of the eye. Such bits of the eye as are visible to their possessor are called 'entopic phenomena'. There are others besides the floaters, as we shall see presently.

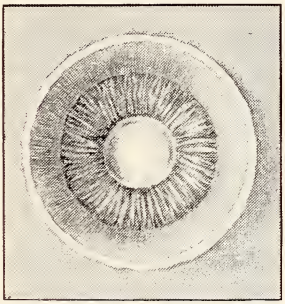
The human retina is not unlike that of the lizard. It has a macula, and the light-sensitive cells are of two sorts, rods and cones. The macula is all made of cones and is opposite the centre of the pupil. As we pass from the macula to the periphery we find fewer cones and more rods. The retina is backed by a thin pigment layer and by a choroid of blood-vessels, but no shiny tapetum. The human retina, like the cat's, has a network of fine blood-vessels on it, and these do interfere very slightly with sight by getting in the way of the light rays. Again, we are so used to seeing them that we are not conscious of them. We can see them entopically, however, in two quite simple ways. First, if we look at a bright sky and find our vitreous floaters and watch them carefully we shall be conscious of a swirling movement of minute bright specks all over the background (except just at the spot where we are looking). These swirling bright specks are the tiny blood corpuscles (cells) running along the network of tiny vessels. There are none just where we are looking, as there are no vessels in the macula, where they would be a nuisance. A way of seeing the vessels themselves (as distinct from the circulating blood in them) is to stand in a dark room looking straight ahead and to shine a small torch or a candleflame

Plate VII



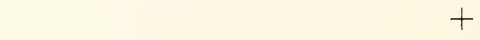


a. Shows a cross section of the eyeball with the various layers and structures inside it. The line drawing above gives the names of the various parts.



b. Shows a cross section taken at right angles to that shown in a. The front part of the eye is shown and is seen from behind. The outer rim of the circle is the cut edge of the sclera. The central clear circular structure is the lens surrounded by the pleated-up ciliary body.

from the side, on to the eye (at right angles to the line of sight). If you move the light slightly you will catch glimpses of a pattern of dark lines like the branches of a tree on a kind of shiny background. These are the shadows of the larger vessels thrown into movement on the rods and cones by the small movements of the light. If you become good at this game you may notice that the branches all appear to come out of a round dark hole just to the side of where you are looking. This is the head of the optic nerve, and the vessels come in through the hole in the sclera through which the nerve goes out. Since there are no rods and cones on the end of the nerve this little bit of the background of the eye is a blind spot. We can easily see that this is so by another method. Stick a long pin through a tiny piece of white paper (about half the diameter of a pea) and hold the pin upright by the other end. Shut your left eye. Look straight ahead at a fixed spot in front of you with your right eye. Hold the paper disc up in front of you and slowly move it horizontally outwards towards the right. If you keep your eye still and do not follow the movement of the paper with it you will find that this disappears for a second or two during the movement outwards. This occurs when the picture of the paper disc falls on the optic nerve. Another way is to look at the dot below



with your right eye, when you will notice that you cannot see the cross, but if you look at the cross with your right eye you can see the spot. The reverse holds for the left eye. This accounts for the adoption of the

two-eyed stance by cricketers. Many a stump has been knocked out because the batsman has 'lost' the ball on the blind spot.

Now we come to a stumbling-block which you may have foreseen. When we make the cross disappear with the right eye it was on the right or outer side of the dot we were looking at. If, however, we look into the right eye of a friend with the little bit of mirror with a hole in it (that we used to look at the cat's tapetum and optic disc and vessels) we shall see, if we are expert enough to find it, that the the optic disc is on the left side of the macula (the victim's left, not the observer's). This is because the right side of the retina sees things to the left, the top of the retina sees the floor, the bottom sees the sky, and so on. In other words, the picture in the eye (on the retina) is reversed. If we look carefully at the image formed by the lens in chapter III we noticed that it also was reversed, and if we look at a ground glass screen at the back of a camera (not the view-finder) we shall see that the picture is again reversed. Therefore, if the optic nerve is on the left of the macula in the right eye, its corresponding blind spot will be on the right of where we are looking, since rays of light coming from the right fall on the left side of the retina. People often think it queer that we 'see things upside down', and have to reverse them in our minds, but this reversal is already made by the readjustment of the message on its way through the brain, and no conscious effort is required to relate things to their right position in the field. Experience is necessary to gauge the relative distance of objects and their accurate direction in space and full stereoscopic vision is

not present at birth, but has to be learned (and actually is not always learned perfectly).

We cannot see, by examining the eye, beyond the head of the optic nerve, neither can we see the ciliary body, as it is hidden behind the iris. The nerve fibres in the optic nerve run through a hole at the back of the bony orbit and link up with various parts of the brain. We can distinguish various levels of linking. At the first level, and therefore unconscious, are the electrical connections which ensure the correct working together or co-ordination of the movements of the eyes and eyelids; for example, the blinking necessary to keep the cornea moist and clean, the sudden protective closure of the eye against foreign bodies or threatened injury, the following of a moving object with both eyes and the convergence and accommodation necessary when we look at a near object. All these movements, as well as the contraction and dilatation of the pupil with altering amounts of light, are automatic and therefore practically tireless, again like the movements of the heart and lungs and of the stomach and intestines, which go on all the time, even in sleep, and in healthy people are never noticed at all. These automatic and reflex actions of our bodies can by introspection be brought into consciousness, and the individual may worry about them and begin to think they are abnormal, unless he understands their nature and that they can be safely left to look after themselves. It is the failure to realize this largely automatic element in the acts of seeing and looking which has given rise to so many popular misconceptions about eyestrain and the use of spectacles.

We can, of course, become conscious of our eye movements if we think about them, and, indeed, in the

best developed eyes there is both a smoothly working set of reflex actions and, as well, a conscious voluntary control of them which can be exercised at will when necessary, but which is usually not thought of at all. With practice most of the movements of the eyes can be controlled by the will.

At the second level of the connections with the brain the object, when looked at, comes into consciousness and we see. At the third, the highest level, we understand what we see, act on it, make judgments about it, remember similar things seen in the past and store up visual memories of it for future use.

. . .

[The following chapters VII, VIII and IX deal essentially with biochemistry and may prove somewhat technical. They may be omitted by readers not interested in this aspect.]

CHAPTER VII

What our Eyes are Made of

The eye is as much a part of the body as, for example, the intestine, and it is therefore dependent on the general blood circulation and on control by the nerves for its maintenance in a useful functional condition. It is a strikingly specialized part of the body, and, because it is specialized, some of its chemical constituents and chemical processes are peculiar to it. But, in general, the chemical processes that go on in the tissues of the eye are thought to be the same as those that occur in other parts of the body.

We shall have to use certain technical terms in this description of the biochemistry of the eye, and in order to make it clearer to those who have no knowledge of chemistry we propose to start by defining, very briefly, some of those terms.

All the diverse stuff in the universe is made up of some ninety elements, substances such as hydrogen, oxygen, nitrogen, phosphorus, carbon, iron and the other metals, etc. Each element behaves as if it were made up of *atoms,* all the atoms of an element being alike. A piece of iron can therefore be imagined to consist of a collection of iron atoms, and a volume of oxygen gas as a swarm of oxygen atoms, all of which are like one another, but which are unlike the iron atoms. Atoms are chemically indivisible and therefore are the basic matter with which chemists work. This does not mean that a single atom can be isolated, weighed and kept in a bottle for future use. Chemists

use collections of atoms—a gram of iron, or a litre of oxygen—and from the way these behave they deduce the properties of the individuals. Elements are seldom present free in the solid matter of the earth or in plants and animals, because they act on each other to form *compounds* which contain two or more elements. A compound is a substance which has a constant chemical composition and constant properties, such as melting point, boiling point, etc., however it may be prepared. Lactose, a kind of sugar that occurs in milk, is a compound, while milk itself is a mixture of lactose and many other compounds. The composition of milk varies, but the composition of the lactose in it remains always the same.

Compounds are made by the action of one element on other elements, or by action between different compounds whereby a chemical change takes place with the formation of new compounds. Such actions may lead to the building up of more complicated substances, or to the breakdown of a complicated substance into several simpler ones. Just as elements behave as if they were composed of small indivisible atoms, so compounds behave as if they were made up of *molecules* which contain the elements in fixed relative proportions. For example, if you decomposed electrically all the water in the Pacific Ocean, or the tiny amount of water in the aqueous humour of your eye, you would find in both cases that two atoms of hydrogen were released for each atom of oxygen. The molecule of water is made like that. It is a relatively simple compound, but some of the complicated stuffs which are present in our bodies contain many atoms arranged in a pattern within the molecule.

Most of the compounds in plants and animals are

made up of the elements carbon, hydrogen, nitrogen, oxygen, sulphur and phosphorus, special compounds containing metals, such as iron, zinc or copper, being exceptional, though essential. We can arrange the compounds which are contained in animals and which they need for their food into a small number of different classes. There are fats such as suet, butter, and lard, and the artificially prepared fat, margarine. There are the carbohydrates which are the starches and sugars, such as potatoes, bread, cane sugar, and glucose, and finally there is the great class of the proteins. Proteins, contained for example in lean meat, eggs or cheese, are more complicated in chemical structure than are the fats and carbohydrates, and are built up from some thirty different compounds called amino acids which are linked together head to tail, as it were, in the protein molecule. These fats, carbohydrates, and proteins are the main constituents of our food, and are converted into the structure of our bodies through the chemical processes of digestion, absorption, and subsequent chemical build-up in all our tissues and organs.

The food we eat is broken down chemically by the various digestive juices of our mouth, stomach and intestine, and the soluble breakdown products, glucose, fatty acids, amino acids, etc., pass through the gut wall into the blood which carries the dissolved foodstuffs round to all the cells of the body. We need food to keep us warm, to keep our hearts beating, our lungs breathing, and our kidneys excreting waste products, and on top of this we need extra food for muscular work, to repair worn out cells and for growth. Apart from our basic foods we must have certain chemicals which are necessary for the regulation of the chemical

processes in the cells themselves, the ultimate users of the food that is brought to them by the circulating blood. So, in addition to the fats, proteins, carbohydrates and salts of our diet, we also require small quantities of special minerals, iron, calcium, copper, zinc, etc., and of other more complicated chemicals called vitamins that we cannot make for ourselves.

When it reaches the body cell the food is either stored for future use, for example, in fat depots under the skin (who has not heard of adipose tissue?), or in carbohydrate stores of glycogen in the liver, or it is burnt up to provide energy or built up into part of the animal body. The chemical waste products for these processes which cannot be further used by the animal are sent out of the body by the lung, kidneys and gut. All these actions are able to proceed at the speed they do because the cells contain certain specialized proteins called *enzymes* which are the catalysts, or accelerators, of all the chemical processes that go on in the animal's body. Enzymes therefore are of the utmost importance. Without them our food would be of no value to us, as we could not digest it or absorb it or use it up to get energy or build it up into new body cells. A great many of the chemical actions of cells are the same in all animals, and therefore we find the same enzymes carrying out these actions in the rat, pigeon, mouse, rabbit, and man, although a mouse enzyme that helps to burn up sugar is not exactly the same, chemically, as a man enzyme that does the same thing. We have said that enzymes are specialized proteins. Some of them are made up of a protein combined with a vitamin, and that is partly why vitamins are so important for us. Like every other constituent of the body, enzymes are gradually destroyed

in use, and so we have to have a constant supply of the parts with which to make them. The protein part we can build up from the proteins of the food, but the vitamin part—of those enzymes that contain vitamins—must be constantly supplied to us ready-made.

Let us follow, for example, the chemical travels of a potato as it is eaten and used by the body. We chew it up, and as we do so the salivary juices get mixed with the fragments and immediately an enzyme gets busy. The starch in the potato combines with the enzyme and the surrounding water, and is rapidly broken down into maltose, a simple sugar. Starch itself is made up of a large number of molecules of glucose, united in a chain like a string of sausages. Maltose consists of only two molecules of glucose, like a single pair of sausages. You can demonstrate this action of the salivary enzyme for yourself quite easily. Take a little starch or cornflour, and make it into a thick paste by boiling up with water. Cool it down to about body temperature, take an egg-cup full, and then spit into this. Shake up this mixture, leave for a little while, preferably in a warm place, and the thick solution will become thin and watery owing to the digestion of the starch by your own salivary enzyme. Starch gives a very deep blue colour when it is mixed with iodine, but maltose does not give this colour, so if you take a drop of your thick starch solution and add a drop of iodine from the medicine chest a blue colour will be produced, but after the salivary enzyme has digested the starch you will find that no colour, or only a dirty brown colour, will be produced when you mix a drop with iodine.

After mixture with the salivary juices the potato starch is swallowed, gets into the stomach, and is

there further mixed with the stomach digestive juices. These have little further action on the starch and maltose—they contain mainly protein-splitting enzymes—and the products are passed through to the small intestine, where other digestive enzymes split the starch and the maltose into glucose. The result of digestion is therefore to turn an insoluble starch into a soluble sugar. Glucose is then absorbed by the cells lining the intestines and diffuses into the blood stream.

The blood from the intestine flows to the liver. What happens now to the glucose? It reacts with liver enzymes and is built up into the kind of starch—glycogen—that animals keep in their bodies, and this is stored in the liver cells. Some of the glucose escapes this building-up process and goes on circulating in the blood, and may reach the muscles, where it is burnt up and provides the necessary energy for muscular effort. One molecule of glucose is burnt up to produce six molecules each of carbon dioxide and water and a certain amount of energy is released. In these chemical processes the glucose molecules are not isolated from all the other chemical processes that are going on in the muscle. They form phosphate compounds, they react with other food breakdown products and with the oxygen in the blood-stream, and they are, as it were, pushed about from pillar to post until gradually nothing is left of them but carbon dioxide and water, and all the energy liberated by these chemical changes has been translated into heat and muscular work. Each chemical change is controlled by a separate enzyme in the muscle cell.

So far as we know, the eye uses up the food in the same way as it is used elsewhere in the animal body, and it will therefore suffer in common with the rest of

the body if we do not get enough food or if our food is not of the right kind. Rolling the eyes around, or changing the shape of the lens, or contracting the pupil, all involve muscular work, and the chemical changes that occur in the muscles of the orbit, ciliary body, or iris are the same as the chemical changes that go on in the muscles of other parts of the body. Description of general biochemistry is not the purpose of this book, and the main point we wish to make clear is that the eye is not an organ that can be taken out, scraped, and put back—but is as closely connected with the rest of the body, both chemically and physically, as one joint of the finger is with the next, and has the same basic chemical methods of obtaining the energy necessary for functional maintenance and repair.

Most of the chemical specializations peculiar to the eye are concerned either with the maintenance of transparency, so that the rays of light may penetrate through the eye tissues to the retina, or with the chemical changes which enable the light energy to be used to send electrical messages along the optic nerve to the brain. Let us consider first the way in which the eye is chemically adapted so that light can penetrate easily to the retina.

The Cornea

Everyone is familiar with the way in which substances change in transparency as they swell; the opaque flakes of dry gelatine turning to a clear jelly on soaking in water, cornflour becoming at least semi-transparent on boiling, and even soap being translucent or opaque according to circumstances. The list can be extended almost indefinitely and all that concerns us here is that the transparency of the eye

tissues is bound up with their degree of swelling in exactly the same way as the transparency of the common household substances we have already mentioned. The early embryo is transparent all through, and the cornea, lens and vitreous body of the eye have retained this embryological state. The cornea, through which the rays of light enter the eye, is a specialized part of the outer coat of the eyeball, which consists, both in the opaque sclera and in the clear cornea, mainly of the protein, collagen. Collagen is chemically related to gelatine and it is also the main constituent of skin, tendon, and the leather of our boots, and like gelatine its opacity or transparency depends on its state of swelling. In the cornea the collagen is arranged in criss-crossing sheets of fibres which make the cornea remarkably tough, and the swelling of these is kept constant partly by the constancy of the salts contained in them and partly by the membranes at the front and back of the cornea. If a cornea is cut out and immersed in water it will swell to about six times its normal thickness, owing to what is called the osmotic, or solution, pressure of the salts and other substances in it, and it will also become semi-opaque to light, for both swelling and shrinking lead to increase in opacity. Water dropped into the living eye does not lead to swelling and opacity of the cornea, because there is a barrier of surface cells (epithelium) between the collagen and the outside water and this layer of cells regulates the intake of water into the cornea.

On the inside surface of the cornea there is another layer of cells (endothelium) which controls the intake of water, salts and nutrients from the aqueous humour, and damage to either epithelium or endothelium leads to unregulated corneal swelling and opacity. The

clarity of the cornea depends on maintenance of the smooth unbroken surface of epithelial and endothelial layers. It is clear from recent experiments that the chemical activities—the metabolism—of these cell layers regulates the swelling of the cornea and so controls the optical clarity. For example, people who wear contact lenses may find that after some hours they become uncomfortable and that vision becomes misty. Experiments showed that when an ordinary contact lens was fitted onto the eye of a guineapig, the cornea became quite noticeably swollen after some hours, but that if a bubble of oxygen was trapped in the space between the cornea and the contact lens the cornea remained unswollen, while the bubble of oxygen gradually became smaller. It therefore seemed probable that, if oxidation processes could continue in the corneal epithelium, swelling would be prevented. This idea was reinforced by finding that the swollen cornea of the guineapig had more lactic acid than normal and less glycogen. Lactic acid is made from glycogen and glucose in the absence of oxygen and does not provide as much energy to a tissue as oxidation of glucose to carbon dioxide and water. If therefore one thinks of the corneal epithelial cells as having to maintain the unswollen state of the cornea by a chemical pump which pumps out unwanted water, then cutting off oxygen by the contact lens can be supposed to cut off much of the energy required by this pump. Present day contact lenses have peripheral holes or grooves to allow free flow of fluid containing dissolved oxygen.

The relation of chemical processes to corneal swelling can also be seen in test tube experiments. The cornea of an ox eye from the slaughterhouse is at first clear and bright but if it is cut out of the eye and put

in saline in the refrigerator it will swell. If this cornea is then warmed to body temperature in air with saline plus glucose it reverses its swollen state and becomes almost clear again. But it will not pump out the excess water unless both air (oxygen) and glucose are present. The processes can be explained in this way. Cooling the cornea stops the enzymic processes of metabolism so that water flows into the tissue by osmosis and is not pumped out by the chemical pumps of epithelium and endothelium. When the cornea is rewarmed these pumps start again so long as the fuel to drive them—oxygen and glucose—is present.

The corneal epithelium has an active metabolism, in spite of having no blood supply. Oxygen diffuses into it from the air, the tear fluid, the blood vessels at the edge, the aqueous humour which bathes its under surface and, when the eyes are shut, from the blood vessels on the inner surface of the lids. Glucose is completely oxidised to carbon dioxide and water or converted to lactic acid. In contrast, the collagenous stroma of the cornea probably hardly uses oxygen at all, does not oxidise glucose but converts it only to lactic acid by chemical reactions which do not need oxygen. There are few cells in the stroma compared with the epithelium. Apart from the fine uniform fibrils of collagen, the stroma contains mucopolysaccharides which are complex carbohydrates found in most of the connective tissues of the body. These substances are gels (clear jellies) and bind the collagen fibrils together. They appear to be important for maintenance of clarity of the cornea.

We are still at the beginning in studies of the connection between metabolism and pathological condi-

tions in the cornea. There are many infections and degenerations of the cornea where no connection between a change in corneal metabolism has been traced but nutritional deficiency gives examples where metabolic change results in corneal degenerations.

Everyone, in this age of unrestricted advertising, knows that the health of the body depends on an adequate supply of vitamins. Earlier in this chapter we stressed the fact that the eye tissues are biochemically similar to the other tissues of the body, so it is not surprising to find that vitamins are also essential for the maintenance of the health of the eye. Professor Hopkins, who published his first paper on these yet unknown substances in 1912, called them accessory food factors. This aptly describes them still. They are chemical substances which most animals cannot make in their own bodies from the ingredients of their food, but which they must have in small amounts in order that they may use such foodstuffs to live and grow. We do not live on them, but we cannot live without them. If we eat a good mixed diet we get all the vitamins in our meals, but anyone living for long on a diet restricted to one class of foodstuffs is liable to become vitamin-deficient.

Riboflavin—vitamin B_2—is one of the substances that man and many other animals cannot make for themselves but have to get from their food. The healthy animal contains riboflavin in all parts of the body as it is an essential part of enzymes that oxidise food-stuffs in the tissue cells. A man needs about 2 mg. a day and such foods as wholemeal bread, liver, milk and eggs supply it. If none is supplied, signs of deficiency soon appear as there are no stores of riboflavin

in the body. Young animals cease to grow and the corneas become invaded by blood capillaries. A similar change in the cornea has been seen in ill-fed and underfed people, first in the Southern States of America and then in the Middle East and India. The eyes become sore, red and watery and very uncomfortable. Examination shows that the cornea is vascularized, the vessels lying just under the epithelium. The corneas of riboflavin deficient animals oxidise glucose less readily than normal and, naturally enough, contain less riboflavin. If the vitamin is added to the diet the cornea becomes less painful within twenty-four hours and the blood capillaries empty within a few days. Quick repair is characteristic of cure of a vitamin deficiency. In this case the enzyme proteins are presumably still present in the deficient cornea and as soon as riboflavin is administered it reaches the corneal cells and can play its part in oxidation, being itself reversibly reduced and oxidized in the process.

Vitamin A is also essential to the cornea. Later chapters describe its role as part of the light-sensitive pigments of the retina. Its role in the cornea is less well understood but that it is essential is in no doubt. All epithelia in the body need vitamin A and when it is absent from the food, the epithelia of the trachea and bronchioles, the vagina and the colon suffer, as well as those of the conjunctiva and cornea. The cells become dry and thin and flake off the surface—they are said to be keratinized like the normal surface cells of the skin. When this happens to the corneal epithelium it becomes rough and opaque and vision is distorted and impaired; the condition is known as xerophthalmia. If the deficiency persists the corneal collagen and mucopolysaccharides of the stroma seem to liquefy

and the cornea ruptures. This leads to blindness as, even if the cornea is repaired, there is a dense scar. The chemical reactions in epithelial cells for which vitamin A is essential are not known with certainty. Recent experiments show that it may catalyse the formation of mucopolysaccharides that have a sulphate group attached to their molecules. Such substances are present in the cornea and it may be that vitamin A is necessary for their formation.

Blindness due to destruction of the cornea in vitamin A deficiency occurs most readily in young animals, babies and toddlers. This is because animals, and man, as they grow, store large amounts of vitamin A in their livers. The young do not have such stores to fall back on and become deficient more readily. Vitamin A is insoluble in watery media but is soluble in fats and oils and the oil from fish livers is particularly rich in it. Cod liver oil has been the traditional vitamin A rich supplement of children's food for many years and liver itself was known thousands of years ago in China, Egypt and then Greece as a cure for eye troubles. It is therefore all the more shaming to realize that blindness still occurs in children due to deficiency of vitamin A, and that once blind they are blind for life. In 1958, Dr. Oomen, a Dutch doctor in Indonesia, could write:

"Xerophthalmia has been the most bitter pill for me to swallow during 18 years of doctor's work in Indonesia. The over and over repeated experience of discovering a child, recently blinded, in the arms of the mother; having to tell her that I now could do nothing more to save its eyesight; remembering that I could have done so with a few spoonfuls of cod-liver oil some days ago; these things still enter my night-

mares. They belong to the most vivid examples of what dis-privileged people in under-developed regions sometimes miss. . . . What on earth is nutritional science good for if, even in the atom age, it is not capable to counteract one of the foulest consequences of bad nutrition? Do you realize, that since the days of Mori, 60 years ago, not in Japan, but in countries like Indonesia, not one step forward has been taken, in spite of a mountain of thoughtful attention paid by doctors?"

Turning now from cases where the metabolism of the tissue controls its normal state to a case where the actual chemical structure of one constituent is of great importance, let us consider the collagen fibres of the corneal stroma. Collagen is a protein; it is present in skin, tendons, bone and connective tissue as well as cornea and sclera and in fact it makes up about one third of the total protein of the body. One of the characteristics of proteins is that they are antigens and if injected into another animal they stimulate formation of antibodies. These antibodies circulate in the bloodstream and tissue spaces and combine with the antigen if it is present. This antigen-antibody reaction can be part of inflammation, the tissue in which it takes place becoming swollen, inflamed and vascularized. Collagen is peculiar in that it does not form antibodies. It is a largely insoluble fibre and therefore it may be difficult for the collagen molecules to reach the antibody-forming cells, but this cannot be the whole story, as gelatine, which is formed from collagen on boiling in water, is also non-antigenic. Its non-antigenicity may be due to the fact that it contains only very small amounts of tyrosine and tryptophan, two amino acids that provoke antibody formation.

The result of this non-antigenicity of collagen is that a cornea—which is 85 per cent collagen—from one rabbit, implanted into another rabbit, does not provoke an inflammatory response. The lump of implanted cornea simply stays put. This is completely different from the behaviour of skin. A skin graft cannot be made from one person to another. The graft will be sloughed off due to antigen-antibody reactions between host antibodies and the antigenic skin proteins of the graft. The skin graft does not take, but a corneal graft from one person to another will take because collagen is not antigenic. Thus, if there is a scar in the cornea obscuring vision it is possible to cut out the dense scarred area and replace with a piece of clear cornea from another person. The reaction between host and graft has been studied in great detail and it seems probable that all constituents of the graft cornea, epithelial and stromal cells, soluble proteins and so on, disappear and are replaced from the host. But the graft collagen remains and forms a well ordered scaffolding to be colonized by host cells. Corneal grafting is an operation now carried out successfully all over the world but it could not be done if collagen were antigenic. The chemical composition of the protein determines the suitability of the cornea for this operation.

The Aqueous Humour

After traversing the cornea, the light rays pass through the anterior chamber of the eye, which is filled by the aqueous humour. This bathes the lens, iris and inner surface of the cornea. Most tissues get the food they need for maintenance and growth from the blood circulating in the capillaries; the food substances such as glucose, amino acids, fatty acids, vitamins and

so on diffuse through the vessel wall, get absorbed by the tissue cells and there burnt up by the cell enzymes to provide energy for other chemical or mechanical processes or built up into more complex substances which are constituents of the cells. For example, proteins are synthesized from amino acids in all growing cells, glycogen may be synthesized from glucose, and fat from fatty acids such as acetic or malic acid. An active tissue cell is one of the most complicated chemical factories in existence but, although an apparent hurly-burly, each chemical reaction is co-ordinated with all the others.

Neither cornea nor lens have any blood supply so that their food must come from another source. The cornea gets some by diffusion from blood vessels at its circumference but most of its nourishment and all of that necessary for the lens must be supplied by the aqueous humour. This fluid therefore plays a very important part in the chemical processes of the eye tissues. In order to do this successfully, it must be constantly renewed, as, if it were static it would soon be depleted and the lens and cornea would starve. This renewal of the aqueous is achieved by a circulatory mechanism whereby the aqueous is formed in the eye, and after bathing the lens and cornea is then drained away out of the eye into the veins. It is produced by the ciliary body, which, as well as containing the circular muscle described in Chapter V, acts as a gland. It lies just at the back and at the base of the iris. The aqueous is drained away through a minute canal that leaves the eye in the angle between the iris and the cornea which means that it must flow constantly over the front surface of the lens, both surfaces of the iris and the internal surface of the cornea.

One of the latest estimates of its rate of flow has been made by seeing how rapidly water that has a different sort of hydrogen, heavy hydrogen, in its molecule, appears in the aqueous fluid after it has been injected into the animal at another point. This particular water can be detected in the presence of normal water owing to its greater density, and therefore if it is injected into the blood-stream and then appears in the aqueous the amount that appears in a given time tells you how rapidly water exchange between the blood and the aqueous takes place. In rabbits this water exchange goes on at such a rate that half the water of the aqueous is changed every 2-3 minutes. This is probably considerably faster than the rate of exchange of the whole aqueous with its contained solids, but clearly indicates that the fluid bathing the lens and cornea is anything but stagnant.

The aqueous contains almost everything that is in the blood except the proteins; there is glucose, a small amount of dissolved oxygen, various inorganic salts and amino acids. It is in fact rather like a finely filtered blood, all the larger substances having been retained by the filtering blood-vessel wall and the simpler substances getting through; but it is not exactly a mechanical filtrate, as there are substances whose concentration in the aqueous is higher than it is in the blood. For example, ascorbic acid, usually called vitamin C, is 20-30 times more concentrated in the aqueous than it is in the blood. A great deal of work has been done on the relationship between vitamin C and the lens; in scurvy, which is the disease caused by lack of vitamin C in the food, the vitamin C content of the aqueous gets very low, but no definite changes have been found in the lenses of scorbutic animals, nor

has the lack of vitamin C been found to have any relation to opacity of the lens, or cataract.

The aqueous has another function besides that of feeding the lens and the cornea, and that is to keep the pressure inside the eyeball at a constant level. If the pressure were not constant the eyeball would either bulge or sag, and focusing the light on the retina would become impossible. The pressure inside the eye is normally equal to that of a column of water 11 inches high, and it is regulated by the constant production of aqueous and its constant drainage away out of the eye through the canal in the angle between the cornea and iris. If the canal gets blocked or if too much aqueous is produced, the pressure within the eye will go up, and if production fails the pressure will fall and the eye will shrink. An increase of pressure is the more usual occurrence, and leads to a disease called glaucoma, which, as you see, may be caused by a failure of any one link in the pressure-regulating mechanism.

A good deal has been said about enzymes and how essential they are for growth and function of the body and the eye. In general the efforts of the doctor are directed to keeping the enzymes active but a real advance in treatment of glaucoma has come from use of an enzyme-inhibitor. Secretion of the aqueous humour by the ciliary body depends at least partly on concentrations of bicarbonate, sodium and chloride ions in the body fluids. If these are lowered, secretion of aqueous humour falls and in consequence the pressure inside the eye will also fall, which is exactly what is necessary in glaucoma. Until recently glaucoma patients were surgically treated so that the aqueous humour could drain away more freely, but in the past

few years many of them have been successfully treated with pills of Diamox, a substance that inhibits the enzyme carbonic anhydrase and reduces the bicarbonate level in the body fluids. The chloride and sodium concentrations in plasma are also reduced at the same time and the effect is to diminish secretion of aqueous humour and so reduce the pressure in the eye.

The Lens

The lens is a fascinating tissue biochemically as well as optically. It is simply a colony of cells suspended in position by the suspensory ligament and unconnected with either nerves or blood vessels, and its clarity, like that of the cornea, depends on the degree of swelling of the fibre-like cells of which it is made up. Embryologically the lens begins as a tiny clump of cells that breaks off from the skin and gets incorporated in the developing eye. At this stage the whole embryo is transparent and the lens retains this embryological property. The lens fibres which are simply elongated cells, are laid down concentrically, like the skins of an onion, on the outside of the original nucleus of embryonic cells, so that during life the lens develops layer after layer of fibres, the newest ones being on the outside, and the oldest being trapped in the centre of the sphere. The whole system is enclosed in a cellophane-like membrane which is not permeable to the lens proteins and so maintains these inside the lens in a definite state of hydration. If this capsule is injured and torn the lens proteins dissolve out, the lens becomes opaque and may, in young people, finally dissolve away altogether. The proteins of the lens are unlike those of any other part of the body, but those from different animals seem to be

exactly the same. They are not closely chemically related to collagen, the protein of the cornea, but are more like the proteins of egg-white. Chemical changes in the lens proteins accompany the opacities of the lens that are called cataracts. The production of opacity in the lens is always accompanied by change in its chemical composition, which will include change in the water and salt content, in the solubility of the lens proteins, or in the permeability of the lens capsule. It is the balance between all these which keeps the normal lens crystal clear.

About 35 per cent. of the wet weight and almost the whole dry weight of the lens is protein. Since the whole lens is derived from a single clump of ectodermal cells the proteins are characteristic of these cells but may vary according to the stage of growth. Thus proteins from the old lens fibres in the centre of the lens may differ from new-formed protein in the cortex. The first point of interest is that lens proteins show organ specificity rather than species specificity. Immunologically, our lens is similar to the lenses of other mammals and an animal can become sensitized or immune to the proteins of its own lens. An inflammatory reaction severe enough to cause temporary blindness occurs rather rarely in the eye after an operation for removal of a cataractous lens and this reaction is thought to be due to sensitivity of the patient to his own lens protein.

The proteins of the normal mammalian lens can be separated into three classes according to their solubility. The young lens contains a small amount of insoluble protein, albuminoid, and two groups of soluble proteins, the α-crystallins which are insoluble in very weak acid, and the β-crystallins that are soluble at

this acidity. New experimental methods using paper chromatographic and electrophoretic methods of separation have shown up to thirteen different fractions of lens proteins. In the growing lens these must all be actively synthesized but perhaps in the ageing lens fibres they are broken down at different rates and so the old lens will have a different protein composition to the young.

The lens is dependent upon oxygen and glucose in the same way as the cornea and in test tube experiments the lens imbibes water and swells peripherally if kept without oxygen and glucose. Again, like the cornea this swelling and hydration is reversed if oxygen and glucose are supplied, showing very clearly that lens structure and clarity depend on its metabolism.

Nearly all the energy required for lens maintenance and growth—the synthesis of proteins from amino acids and so on—comes from breakdown of glucose, and almost all the glucose is broken down to lactic acid. This is a process that does not need oxygen, but a small part of the glucose used is directly oxidized and this presumably is the process that makes oxygen essential for the lens.

Increasing hardness, or, to put it another way, decreasing flexibility of the lens with loss of focusing power is probably the result of the continuous growth of the outermost lens fibres with compression of the ones in the centre. But the compressed lens fibres remain optically clear. Senile cataract, the opacity of the lens that afflicts to a greater or less degree many elderly folk all over the world is much more than a hardening of lens fibres. We know nothing of the causes of this form of cataract, but, judging from other

cataracts, we can predict that it is triggered off by a change in metabolic activity of the lens. This may be due to a change in the lens itself or due to a change in the supply of food through the aqueous humour.

Study of experimental cataracts has given interesting information on the importance of lens metabolism for its clarity. If a young rat is made diabetic, cataracts develop. Diabetes is a disease in which the metabolism of glucose is upset and as glucose is the main fuel of the lens cells it is not surprising that the lens becomes abnormal in the diabetic animal. Such cataracts occur rarely in young diabetics before they are given insulin to regulate their sugar metabolism. If they do occur they can often be reversed by insulin dosage.

Cataracts also occur in young rats which are fed large amounts of xylose or galactose, two sugars that are not used chemically very readily by body tissues and so accumulate in the blood stream. The galactose cataract of young rats is paralleled by the cataracts which occur in babies who cannot metabolize the galactose of milk. Milk contains lactose, a sugar which is broken down in the body to equal parts of glucose and galactose. Nearly all babies can use both these substances but a very few do not have one of the enzymes necessary to metabolize galactose. These babies accumulate galactose in their blood, they do not grow and cataracts develop in their lenses. This lack of enzyme is an inherited defect and all grades of severity are found in relatives of affected babies. When food containing no galactose, which boils down to a non-milk diet, is given to galactosaemic babies they start to thrive and their cataracts may get better. As they grow, some of the missing enzyme may appear so that in later life milk

is not so toxic. Kalckar has shown that the enzyme missing from the blood and tissues of babies that develop galactosaemia is concerned with change of galactose phosphate to glucose phosphate, but in spite of this enzyme lack there is little accumulation of galactose phosphate in the tissues either of babies or of young rats fed excessive amounts of galactose. Dr. Ruth van Heyningen in Oxford has found that the galactose-fed rat does not oxidise galactose but actually reduces it to the sugar-alcohol, dulcitol. This substance then piles up in the lens even before the cataract develops. The diabetic rat which has a high blood glucose and the xylose-fed rat also show this change in sugar metabolism and reduce the sugars rather than oxidizing them. The sugar-alcohols sorbitol and xylitol accumulate in the lens after a few days of the experiment and then the cataract develops.

The Vitreous Body

Between the lens and the retina lies the vitreous, which completely fills, as you might say, the body of the camera, and is the last stage on the path of the light rays to the retina. It is made up of 99 per cent. water with only 1 per cent. of solid matter, yet it is far more like a solid than a free-flowing liquid, and if it is shelled out of an eye it retains its shape, looking very much like one of the small jellyfish one can pick up round the coast. Two substances in the vitreous give it this extraordinary viscosity and rigidity. One is a starch-like substance, or polysaccharide, called hyaluronic acid, which can be extracted from the vitreous and purified and when redissolved will again form a viscous treacly solution. Hyaluronic acid occurs also in the jelly of the umbilical cord and the joints, but the

nearest analogue is frog spawn jelly. In this case the jelly protects the egg from the buffets of the environment, just as the vitreous jelly maintains the retina in position and helps to control the shape of the eyeball. The other substance which holds the vitreous together is collagen. Very fine collagen fibres permeate the whole mass and hold the swollen hyaluronic acid gel within the meshwork. The vitreous body can easily be separated into its two parts by filtration or centrifugation. The viscous hyaluronic acid solution separates from the small fibrous residue of collagen fibres. In the eye the vitreous body is anchored particularly firmly to the outer coats of the eyeball in the area of the retina just behind the lens. Here, rather coarser fibres springing from the retina, thread through the outer layers of the vitreous body.

How can one relate this structure to the behaviour of the vitreous body during life? Two troubles which affect vision are partial liquefaction of the vitreous body and the more serious trouble of retinal detachment. When you look at a blank wall or a clear sky you frequently see small floating grey or black dots or threads which, if your eye is still, gently move across your field of vision. These "floaters", as they are called, are present in nearly everyone's eyes and are little tags of fibres in the vitreous body. They move because the vitreous body is not rigid. They move very slowly and only for small distances because it is a viscous jelly. These small fibrous knots and remnants are normal, but if the fibrous network of the vitreous body gets disorganized, either through its own partial liquefaction or through change of the viscous gel of hyaluronic acid to a fluid state, then serious opacities may develop.

A still more serious threat to vision is detachment of the retina. Here a part of the retina tears or floats off and so becomes useless as a light-receiving mechanism. Some types of retinal tears and detachment may be due to traction on the retina by vitreous fibrils. The retina is most firmly attached to the vitreous body at its periphery just behind the lens and this is the area where retinal degeneration and detachment most frequently take place.

The Retina

Further chapters describe the chemistry of the light-sensitive pigments in the retina, here we are concerned with the retina apart from these. The retina is a part of the brain and just as the brain has the most intense metabolism of any part of the body so the retina has the most intense metabolism of any part of the eye. If this metabolism is stopped by a short interruption of the blood supply, blindness may result. The glucose in the blood capillaries is rapidly absorbed and either oxidised completely to carbon dioxide and water or broken down to lactic acid. Many attempts have been made to find changes in the retina, other than changes in visual pigments, that are the direct result of illumination of the tissue. So far there has been no success, but a study of the effect of chemical poisons shows that if metabolism is upset, vision is impaired. For example, iodoacetate, a substance that will inhibit the activity of several enzymes concerned with glucose breakdown, upsets retinal metabolism and causes degeneration of the rod cells. A single injection of iodoacetate into an experimental animal immediately abolished the electrical response of the retina to light and inhibited breakdown of glucose. The light-sen-

sitive rod cells of the retina then degenerated gradually over the next few weeks. The picture of the iodoacetate poisoned retina was reminiscent of the picture of the retina in retinitis pigmentosa, a human disease where progressive degeneration of rod cells takes place.

A perfect example of change in retinal metabolism leading to blindness has been demonstrated in retrolental fibroplasia, a disease produced in premature babies by too enthusiastic use of oxygen therapy. This disease now no longer exists, but the story of its investigation is fascinating and, as it illustrates the methods by which experimental work has to be done when only human material is available for study, perhaps it is useful to set it out here in some detail.

First of all the disease is noticed. It only occurs in the tiniest of the premature babies and it is only really noticeable some time after birth. Blind eyes are removed at operation and studied microscopically and it is found that the retina is folded up and that from the retina come fibrous strands which unite to form an opaque membrane behind the lens. How did it all start? Was the disease really present at birth but not really noticeable, or did it only begin after the baby was born?

This point was settled by careful examination of the eyes of all premature babies weighing 3 lb. or less from the time they were born. It was found that their eyes were quite normal at birth and showed no sign of abnormality for three-five weeks afterwards. After that they might still remain normal, but in some babies the retinal blood vessels got wider and more twisted and the retina itself swelled and sent off fibrous bands into the vitreous body. Finally the fibrous bands unit-

ed into a membrane behind the lens and at the fourth or fifth month of life the process stopped developing but the baby was more or less completely blind.

The disease therefore is a disease developing after birth. Could it be due to faulty management of the baby's food or environment? It is pretty difficult to find a suitable diet for these tiny babies and considerable care had already been spent on evolving one which they could take. They don't manage fat well so it was a high-protein and low-fat diet; vitamin C was added (against scurvy), vitamin A was added (growth and eye maintenance), vitamin D was added (bones), vitamin K (haemorrhage) might be given and finally iron was added for blood formation. At least such was the practice of the "best" hospitals, and curiously enough the babies seemed to fare worse in these than in the poorer hospitals where less care was taken. In these best hospitals also the babies lived in incubators and the atmosphere they breathed was kept very rich in oxygen as there is a tendency for these tiny babies to breathe so weakly that they turn blue from anoxia.

The first investigation seemed to point to a deficiency of vitamin E being the cause of the retinal malformation but then in 1951, Dr. Kate Campbell of Melbourne reported that babies kept in incubators and supplied with a high concentration of oxygen were more likely to develop retrolental fibroplasia than those given oxygen only as and when it was necessary. This was fairly soon confirmed from other hospitals in different parts of the world and then to clinch the matter, somewhat the same kind of retinal overgrowth and malformation was produced in baby mice and kittens by keeping them in an atmoshere of oxygen. It is only the developing retina that is upset by oxygen in this

way; the retina of adult animals or of man is unaffected. Thus in the span of a few years this disease—the direct result of efforts to keep premature babies alive—arose, was investigated and understood, and disappeared, because oxygen is now used only when really necessary.

People who habitually smoke and drink heavily sometimes find their eyesight failing. They cannot see to read even with spectacles although they can see well enough to get about either during the day or at night. Examination of their eyes shows that the central area of the retina, just where vision is most acute in the normal eye, has stopped functioning, although the peripheral area of the retina is normal. Most heavy drinkers are also heavy smokers but sometimes tobacco alone causes changes in vision of the same kind as those caused by excessive alcohol. Cigarettes do not matter, unless they are rolled with shag or very strong tobacco. It is the heavy pipe and cigar smokers who suffer. This change could be due to a toxic effect on the retina and optic nerve of something in tobacco or in alcohol, or it could be a nutritional deficiency. Heavy smokers and drinkers do not eat much. If drinking and smoking are given up before degeneration in the optic nerve has gone very far, the eyes will gradually recover with no other treatment. If the patient stops drinking he usually starts eating because he needs the nourishment, which formerly was supplied by his alcohol intake.

Alcohol amblyopia has also been cured by giving large doses of vitamin B, while drinking was allowed to continue. One hospital in America collected ten cases of alcohol amblyopia and offered free board and lodging and as much drink as the patients wanted if the re-

quired vitamin tablets were also taken. All the patients accepted the offer and all were cured except for one very advanced case. Several different vitamin tablets were given and it was found that those containing vitamin B_1 gave the best results. It must have been a gay time in the hospital as the average intake of whisky per man was about a quart a day. But the experiment was obviously worth while because it showed that the eye trouble is related to a nutritional deficiency and can be cured by vitamin B_1 while drinking goes on.

During the war some of the prisoners in Japanese hands developed a disease similar to Wernicke's encephalopathy, which has been known for a long time but had always been thought to be due to chronic alcoholism. But the prisoners of war had no alcohol available. There were fifty-two cases among a single group of British prisoners captured at Singapore, the predisposing causes being dysentry and starvation. The main lesions are in the brain but the eyes also suffer. Early in the disease the eyes cannot be kept still but keep up constant small flickering movements. Later, some of the eye muscles become paralysed so that the eyes cannot move in a balanced and co-ordinated manner, the lids droop and it is possible that the retina is also affected. Many different treatments were tried in efforts to restore the prisoners to health as quickly as possible; good food and plenty of it, rest in bed, vitamins of all kinds. Gradually it became fairly clear that in this trouble again vitamin B_1 was the most important vitamin and, if it is injected, the muscle paralysis and eye flicker, or nystagmus, improves within forty-eight hours. Another example of the rapidity of action of a vitamin.

There is yet another type of eye disease which may be caused by lack of one or more of the B vitamins, or by some poison in the food. It turns up in people who are already in a poor state due to starvation and disease. The end result is rather like that of alcohol amblyopia, that is, the sufferer cannot see to read but can get about unaided. Vitamin B_1 however does not cure.

The trouble was first noticed in Africa among school children. Many children leave their home villages and go to live near their schools. They may get a money allowance to buy their own food or they may live and get their meals in hostels. In some places the children are very badly fed (shades of the old English boarding school) and after a few months their eyes are affected as well as their general state of health. It is interesting that home food seems to keep the children healthy and the disease occurs almost entirely among children who are away from their parents.

The main symptom is a progressive blurring of sight. There are graphic descriptions of boys who, after winning scholarships in their younger days, are unable to finish their schooling as they can follow the lessons only by ear, being unable to see the blackboard. This blurring of sight seems to be due to a partial block on the optic nerve so that, just as in alcohol amblyopia, the messages from the light-sensitive cells of the retina cannot reach the brain. One might suppose that this African disease was again a vitamin B_1 deficiency, but as it is not cured by this it cannot be so. The eyes get better if the children are given fairly large amounts of yeast, or yeast preparations like Marmite, which contain other members of the vitamin B complex.

Very much the same kind of blurring of vision and inability to see clearly enough to read affected many prisoners of war in the Far East. It was called 'camp eyes'. It seems to be a nutritional disease but the exact cause and so the exact cure is still uncertain. Sometimes it seems that B vitamins will cure it but sometimes they seem ineffective. In the early stages it is certain that plenty of good food and rest will restore sight, but there are many men today all over the world who did not escape from the Japanese prison camps in time and so far as is known they will remain partially blind for the rest of their lives.

These men suffered from everything; bad food, perhaps poisonous food, and too little of whatever food there was. There was dysentery, beri-beri and starvation. The reports published by the camp doctors after they were released show the resourcefulness and courage of men under duress. Pathological laboratories were built, visual fields were plotted, sections made for histological examination and somehow microscopes constructed. One doctor, who himself suffered from this failure of vision, first noticed it while looking down his microscope. He found that he could not see the red stain in the tissue section he was examining. Now, however much we may be sad or angry that this doctor had 'camp eyes', we must be enormously grateful for this fascinating observation. For this might be an indication that vitamins can affect colour vision.

CHAPTER VIII

The Retina and Visual Purple

The belief that things are seen because some rays or emanations emerge from the eyes and impinge on outside objects was a fairly general one in Greek times, and, indeed, persisted into the late Middle Ages, owing to the reverence felt for the teaching of Galen. The optic nerve was thought to transmit a spirit or 'pneuma' to the lens by way of the retina, and this spirit enabled the lens both to emit and to receive light rays and function as the chief organ of sight. Even Leonardo da Vinci assumed that 'the eye transmits its own image through the air to all objects which are in front of it, and receives them into itself, on its surface, whence the understanding takes them and considers them'. And one may speculate on the exact meaning of these lines from Donne's poem, *The Ecstasy:*

> Our hands were firmly cemented
> With a fast balm which thence did spring,
> Our eye-beams twisted and did thread
> Our eyes upon one double string;
>
> So t'intergraft our hands, as yet
> Was all the means to make us one.
> And pictures in our eyes to get
> Was all our propagation.

At the end of the sixteenth century accurate observation became both popular and possible, and Kepler, the great astronomer and mathematician, on the basis of the work of the anatomists of the time, gave an al-

most complete explanation of the way in which the rays of light are bent within the eye and how this results in the formation of an image on the retina. He thus broke completely with the idea that the lens is the chief light-receiving body, and with that of the emanation of rays from the eye, and substituted the conception of light-rays impinging on the eye from the external world and being focused by the lens on the retina. A few years later, Scheiner, a Jesuit, demonstrated experimentally the formation of an image on the retina by removing the opaque coats at the back of an animal's eye and leaving the semi-transparent retina as a screen on which to catch an image of whatever is placed in front of the eyeball. Newton thought that light rays 'cause a sensation of light by beating and dashing against the bottom of the eye, something after the manner that vibrations in the air cause a sensation of sound by beating against the organ of hearing'. The retina was therefore established as the receiver of light, but the problem of the transmission of this stimulus to 'the understanding' remained untouched.

We now consider that vision consists of several distinct processes which are used separately or together according to the light that stimulates the eye. There is light perception, with ability to perceive differences of light intensity without distinction of colour. There is colour perception, a specific reaction of the eye and brain to light of different wavelengths, and finally there is the perception of the position in space of the thing seen, which is bound up with binocular stereoscopic vision. These processes must all be related in some way to the anatomical and chemical structures of the retina, and detailed microscopical and chemical

studies of the retinas of a large number of different animals have been made in an attempt to explain the complexity of the visual process.

Microscopic examination led to the discovery that the retinas of most vertebrate animals contain two special types of cells, the rods and the cones, and it was suggested that these cells, having a different structure, have also a different function. This suggestion, first made in the mid-nineteenth century, has been borne out not only by a study of the distribution of rods and cones in the retinas of night-loving and day-loving species, but also by chemical investigations and by studies of the nerve connections between the rods and cones and the optic nerve and the brain. In earlier chapters we have assumed the correctness of this idea. Here we wish to give the main evidence on which it is based.

Schultze (1866) found that animals whose retinas contained mainly cones were active only in daytime whereas those whose retinas contained mainly rods were nocturnal in habit.

Microscopical anatomists now agree that examination of a thin slice or section of the retina will give a good idea of the habits of the animal involved. This statement is based on the theory just mentioned, the Duplicity Theory as it is called, that rods and cones have different functions. Rods are mainly concerned with simple light perception, which would be most useful to the animal in dim light and cones are used for acute vision in bright light and for colour perception. To put it simply, you will use your rods by moonlight when you distinguish only shape and distance, particularly of moving objects, but not colour, and you will use your cones by day to see clearly and

to see in colour. Examination of the retina of nocturnal and diurnal animals bears this out. Consider the birds: in general they sleep at dusk and get active at daybreak and their retinas contain predominantly or exclusively cone cells. The owl, that notable exception, has also a notable retina in that it contains large numbers of rods, and if our theory is correct it will use these rods during its night-time hunting. Most reptiles, such as snakes, turtles, and lizards, have pure cone retinas and here again the animals are day-loving. The mammals, in general, have mixed retinas containing both rods and cones, but the relative numbers of the two types vary enormously. For example, cats, rats, and mice, predominantly nocturnal animals, have largely rod cells in their retinas. Man—not so good in the dark—has about twenty times as many rods as cones, but has a special area of the retina, the macula, covered exclusively by cones. Most of the great apes are like man and have mixed retinas, but some lemurs and one monkey, the owl monkey, are nocturnal in habit and have retinas made up of rod cells only.

You will remember that, in earlier chapters, sight was explained in terms of a television set with the retina as the battery of photo-electric cells and the brain at the receiving end. Obviously the connections between the two parts are of great importance for the clarity of reception. Microscopical sections of the retina dyed with special stains show that many rod cells may be connected with one nerve cell and fibre while cones, particularly the macular cones, are far more exclusive and tend each to have connection with a single nerve cell and thus a private wire to the brain. If we consider the effect of this arrangement on vision we can see that rods will transmit a light stimulus in a

rather fuzzy way, since many rods, covering an appreciable area of the retina, are all connected with the same nerve cell which will add together the light stimuli received by the individual rods. Each cone, on the other hand, will transmit a separate message to the brain, and this will give a very clear, sharp impression. For, just as the fineness of detail visible on a photographic plate depends on the fineness of the emulsion of silver bromide on it, so the detail of the retinal image and the impression which reaches the brain will depend on the size of the light-receiving cell and on the individuality or complexity of its connection with the brain (Plate VI).

So much, then, for the evidence presented by comparative and microscopical anatomy concerning the properties of the light-receiving cells of the retina. It all seems in favour of the view that the two types of cells have separate functions in the eye. Such microscopical studies can tell us that the rods and cones have such and such nerve connections with the optic nerve and brain, but they cannot tell us whether a light shining on the retina has any effect on those nerve cells and fibres. Yet, if vision is due to the stimulation of the rods and cones by light and the transmission of the stimulus along the optic nerve to the brain, it is obvious that an analysis of the way in which the nerve fibres react to retinal illumination should help us to unravel the different processes involved. Such an analysis has been attempted, using an electrical technique. When a nerve which is connected to a muscle is stimulated either mechanically or electrically, the muscle contracts, owing to the conduction of the stimulus along the nerve fibre. The conduction of the impulse along the nerve is accom-

panied by a wave of change in electrical potential, and if electrodes, attached to a suitable amplifying and recording device, are attached to the nerve, then the change in electrical potential accompanying the passage of the impulse may be recorded. Conduction along the nerve fibres connected with the retinal rods and cones has been demonstrated in this way. A single nerve fibre, indeed, was separated out from the optic nerve of the King Crab, which has very simple nerve connections with the light-sensitive cells in its eye, and electrodes were attached to this single fibre. A light now shone on the eye caused a regular sequence of uniform electrical charges to travel along the nerve. If the intensity was increased, the frequency of the electrical charges increased also, showing that stimulation of the retina by light is directly related to conduction of impulses along the optic nerve. In the King Crab there is only one type of nerve fibre and this gives a simple response to illumination; but in vertebrate eyes different types of nerve fibre have been distinguished according to the kind of electrical response they give when the retina is illuminated. Presumably each type plays a different part in vision.

We see, therefore, that there is experimental proof that light so affects the retina that electrical impulses are fired off along the optic nerve to the brain. The question of how the light energy is transformed by the retina causing a change in electrical potential has been partly answered by study of the chemical changes that are produced by light in the retinal cells. The idea that sight is ultimately due to the effects of light on some chemical substance or substances in the retina was brought to the fore by the discovery of photography. Silver bromide, on the photographic plate, is

darkened by the light that falls upon it, the darkening at any one point of the plate being proportional to the intensity of light falling on that point. How simple to apply that idea to a consideration of the retina and suppose that a light-sensitive substance in the retina is changed according to the amount and kind of light that reaches it, and that this chemical change causes a change in the electric potential of the connecting nerve cells which is transmitted to the brain, resulting in the subjective sensation of vision.

The first experimental evidence in favour of this idea was produced eighty years ago, when it was found that the pink colour of a retina, freshly removed from a frog's eye, bleaches when it is exposed to light. The pink substance was then extracted from the retina and again the colour of the extract faded when the solution was brought into the light. This light-sensitive substance was called visual purple or rhodopsin, and it was hoped that it would explain the chemical basis of vision. So it has, in part, but in part the chemistry of sight is still unknown. Visual purple is found in the rod cells of the retina, but not in the cone cells. It cannot be extracted from the retinas of those animals (e.g. many birds and reptiles) that contain only cones; yet such animals may have very acute daytime vision. Nor can it be extracted from the centre of the macula of the human retina where there are no rod cells. At present, search is still being made for a substance or substances other than visual purple to account for the light-sensitivity of the cone cells.

The amount of visual purple extractable from the retinal rods depends very much on the state of the animal that is being investigated. If it has been kept in the complete dark for an hour or more, and all

the work of extraction is also carried out in darkness then the yield of visual purple will be good. But if the animal has been exposed to very bright light, the yield will be poor, and instead of getting a pink extract which will bleach at once when exposed to light, you will get a dirty yellow extract that slowly pales on further light exposure. When the eye is taken out of a frog that has been kept in the dark and a light shone into it, the rose colour of the retina quickly fades, but if the eye is then put in the dark it will regenerate most of its original pink colour, thus carrying out, apart from the intact living animal, the chemical process which is considered to take place during life. When a photograph is taken, the plate is spoiled for further exposure, as we must all have found at one time or another, but the 'plate' of your eye builds up a new light-sensitive surface after each exposure, so that vision can be a continuous process. It is possible to over-expose your visual purple—people speak of being blinded by a flash of light—but after a few seconds with eyes shut the retinal enzymes have re-made sufficient visual purple for you to see again.

The accumulation of visual purple in the rods in dim lights is the chemical basis of dark adaptation, the increase in ability to see that takes place when one stays in a dim light for more than a few minutes at a time. On first going into a darkened cinema or out of doors in the country on a dark night one stumbles about in a half-blind state, but after a few minutes the light seems quite sufficient to see one's way. It is not the light that has changed but the light-receiving mechanism of one's eyes. During dark adaptation the human eye becomes about ten thousand times more sensitive to light than it is when light-adapted, and a

fully dark-adapted eye can appreciate a smaller light than any known photo-electric machine.

Visual purple is not bleached to the same extent by lights of all colours. Light must be absorbed by a substance before it can produce a chemical change, the energy of the absorbed light being transformed into chemical energy. A black substance absorbs light of all wave-lengths and transmits or reflects very little light at all. A white substance reflects light of all wavelengths, a red substance absorbs wave-lengths other than red, and so on. Visual purple is a red substance, and does not absorb and cannot be bleached by red light, and in dim lights red colours appear black to us for this very reason. But visual purple does not absorb all the rest of the light, apart from red, equally strongly. By means of the aborption spectrometer, an apparatus which will measure exactly the wave-length of the light absorbed by any particular substance and also measure the intensity of the absorption, so that you get the absorption spectrum of the substance, visual purple has been found to absorb greenish light most strongly, and to be most readily bleached by this kind of light. Now, if we see in dim lights because of the light-sensitivity of visual purple, our eyes should be most sensitive to the green light that bleaches visual purple most readily. This is indeed the case, the light which changes visual purple most readily is the light we can most easily perceive.

Visual purple can be extracted from the retina by solutions of digitonin, bile salts or detergents but is almost insoluble in water. Nearly half of the dry weight of the outer part of the rod may be visual purple and this must form a part of the structure of the cell. Electron-micrographs of rods show that they are made of

piles of discs, rather like the plates in an electrical condenser, and the visual purple is oriented within these discs.

Visual purple has not been obtained in a pure state but it seems to be a compound of protein, fat and retinene, which is the aldehyde of vitamin A. So long as solutions of visual purple are protected from light the red colour of the pigment is stable but when exposed to strong light it bleaches and free fat and retinene are split off from the protein. Enzymes in the retinal extract may then reduce retinene to vitamin A. Wald and Hubbard in Boston have shown that the molecule of retinene has to be a certain shape in order to combine with the protein to form visual purple. The molecules of both retinene and vitamin A ($C_{20}H_{29}OH$), exist in different shapes according to the way in which the different atoms in the molecules are joined to each other. Only one of the shapes of the retinene molecule will form visual purple. When light falls on the pigment, Wald and Hubbard consider that it alters the shape of the attached retinene from a curiously twisted to a straight form, and that, as the attachment of the straight shaped retinene to the protein is very weak, the compound then breaks down and free retinene is liberated, even in the dark. This retinene is then reduced by retinal enzymes to vitamin A and, in order to build up visual purple again, the molecule must be re-shaped to the twisted form before combining with the protein. Even in solution in a test tube, visual purple can be bleached and then re-formed if retinene of the right shape is present. But we still do not know exactly how this combination takes place.

Some very interesting experiments have been done with frozen solutions of visual purple. When these are

exposed to light they hardly change colour at all but when warmed, even in the dark, bleaching occurs. This confirms that light only produces a small change in the structure of visual purple but that this change makes it unstable. It is presumably this first change produced by light that triggers the nerve impulse from retina to brain. How far the bleaching process proceeds in the retina of the living animal is not known. The living eye contains over four-fifths of its full complement of visual purple even in ordinary daylight, but in spite of this the sensitivity of the eye has fallen to a low level. Very small changes in concentration of visual purple cause great changes in sensitivity.

Most of the early experiments were made with solutions of visual purple or by examining the eyes of animals under different conditions, but recently Rushton in Cambridge has worked out an apparatus whereby he can measure the concentration of visual purple in the human eye during life and study how it changes under different conditions of light and darkness. This apparatus is based on the ophthalmoscope (see p. 71). It is well known that the back of the eye, the fundus, may be viewed with an ophthalmoscope, the retina and retinal blood vessels being seen by the light reflected from these layers and from the underlying choroid. When light falls on the visual purple of the retina, some will be absorbed, so that the reflected light will be minus that part absorbed by visual purple. It is possible to measure the light absorbed (the difference between the incoming and reflected light) in a reflexion densitometer and so to measure the concentration of visual purple in the living eye. Rushton has found that in life, as in the test tube, bright light bleaches the pigment in the retina and that this

is reformed in the dark in about thirty minutes. These experiments confirmed that the pigment is bleached most readily in the eye by a green light of wavelength 500$\mu\mu$, to which visual purple is most sensitive and it is present only in those parts of the human retina that contain rods. That is, it is not present at the optic disc or the fovea. These experiments also confirmed that the sensitivity of the eye to light is not simply related to the amount of visual purple that is present. A flash of light which bleached only about one per cent. of the visual purple diminished the sensitivity to light manyfold and the last one per cent. of visual purple to be restored during dark adaptation appears to be necessary for full light sensitivity of the retina. Further experimental work is necessary to understand this.

The chemical relationship between visual purple and vitamin A was first suspected when it was found that rats, which had been fed on food which did not contain vitamin A, had little visual purple in their retinas. If such animals were exposed to bright light, in order to bleach completely any trace of visual purple remaining, they did not regenerate visual purple at all quickly when they were put in the dark. In normal animals, and people, the visual purple in the retina has reached its full level after about thirty minutes in the dark, but vitamin A deficient animals may take an hour or more to build up even a small amount. This has been found experimentally by estimating the amount of visual purple in extracts of retinas of vitamin A deficient rats after varying times in the dark. In other words, the vitamin A deficient animals cannot adapt themselves to the dark. They are night-blind.

Tests of the rate at which the light-sensitivity of

the eye increases in the dark have been used to determine whether people are deficient in vitamin A. One such test of dark-adaptation is carried out as follows: The subject of the test is brought into a pitch-dark room and is then exposed to a bright light, so that most of the visual purple in the retinas is bleached. The light is then turned off and he is shown a very small red light, which he can see with his cones. He watches this steadily and is tested to find how dim a white light he is capable of perceiving with his rods. This light appears a short distance away from the steady red light which he is watching. At first he can only see a relatively bright light, as he has very little visual purple in his retina, but after a few minutes in the dark he will be able to see quite dim lights, and after half an hour or so, when his visual purple is fully restored from the preliminary bleaching, he will be able to distinguish a very small light indeed. Incidentally, it has been estimated that a light can be perceived by the human eye when it is sufficiently bright to bleach five to fourteen molecules of visual purple. Good night vision is certainly dependent on an adequate supply of vitamin A in the food, though in individual cases other factors may also be involved.

Vitamin A is present in liver, eggs, milk, butter and cheese and we can also make it in our bodies from the red pigment carotene present in carrots, tomatoes and many other vegetables. It is stored in the liver in all animals. During the war of 1939-45 a group of young men and women in England volunteered to live on food entirely lacking in vitamin A and carotene in order to see how rapidly they would become night-blind and if other damage to their eyes or other parts of their bodies would develop. It was extraordinary to find that

most of them, even after two years on this food, were still not complaining of night blindness. Tests of dark-adaptation (see p. 128 et seq) showed that as time passed their capacity to see dim lights got progressively worse but it did not get so poor that it was noticeable to the young men themselves. The carotene of their blood fell to zero very quickly and the blood vitamin A fell also but seldom more than to about half the normal level. Apart from the changes in dark-adaptation no other changes were seen. This experiment showed therefore that, in England, young adults had a store of vitamin A in their livers that was sufficient to last them for about two years. Yet vitamin A deficiency still causes blindness in other parts of the world (see p. 102).

So far as we know at present, the rod cells of the retinas of mammals and of some birds, reptiles, amphibia and marine fishes, contain pigments related to visual purple in that the same retinene is released when the pigment is bleached. But not all show maximum absorption of light of 500$\mu\mu$ wavelength, the green light most strongly absorbed by the visual purple of man, cattle and frog. Deep sea fishes have golden pigments which absorb blue light of 480$\mu\mu$ most strongly. This may be an adaptation to life in the deeps where blue light penetrates more readily through the water than light of longer wavelengths. Light penetrating the ocean to 200 metres has a maximum at 475$\mu\mu$ and at this depth the spectral band remaining is quite narrow. It is also possible that the visual pigment of deep sea fish with maximum sensitivity around 480$\mu\mu$ is specially adapted to receive the light emitted by other deep sea animals. The sea deeps used to be thought of as cold, dark and silent but ex-

ploration has shown them to be more like a fun fair on Saturday night with considerable noise—prawns gnashing their whiskers and claws—and bright flashing lights emitted by the animal inhabitants. This light tends to be bluish or blue-green and matches beautifully the maximal absorption of the visual pigments of deep sea fish. This is an obvious advantage for a fish that hunts its food by sight.

The eyes of other marine fishes contain pigments maximally sensitive to light of wavelength 520$\mu\mu$, a green with a trace of yellow in it. There is a great diversity, and, as all these pigments release the same retinene when they are bleached, it is assumed that the difference between them lies in the structure of the protein to which the retinene is attached.

Some fishes and amphibians have visual pigments more violet than visual purple and these have been found to contain a different retinene derived from vitamin A_2 which is slightly different chemically from the more usual vitamin A_1. Wald has studied the distribution of visual violet in fishes and amphibia and considers that it is the spawning environment that has, during evolution, determined which visual pigment is present.

The spawning environment of animals is fixed, the environment in which they live as adults can vary. Many fish either go downstream or upstream to spawn and as adults can tolerate both sea and fresh water. Frogs and newts spawn in fresh water but live on land until they return to water to spawn. The land to the frog is like the sea to the salmon.

Visual violet is characteristic of fresh water fish; visual purple of the sea fish. The retina of the salmon that spawns in fresh water but lives in the sea contains

both, but visual violet predominates. On the other hand, the retinas of eels that spawn in the sea contain predominantly visual purple with some visual violet. The axoltl which is the permanent larval form of the tiger salamander has visual violet in its retina throughout life. The sea lamprey on its way upstream to spawn has visual violet, but on its way downstream after spawning has visual purple. Here the biochemical change seems to precede the change in environment.

Crescitelli made the very interesting observation that tadpoles of a toad contained visual purple whereas tadpoles of a Pacific tree frog taken from the same pond contained visual violet. The adults of both species contained visual purple. Tadpoles of ordinary English frogs also contain the adult visual purple system; one is therefore left with the idea that though change in visual pigment frequently takes place on change of habitat and metamorphosis from larval to adult form, this is not essential to the process of emergence from water to land. One must also remember that is is possible to change a rat into a freshwater fish, visually speaking, simply by feeding it on vitamin A_2, rather than vitamin A_1. After some weeks the retina of the rat contains predominantly visual violet. The predominant visual pigment depends on the food as well as on evolutionary history expressed in spawning environment.

The eyes of various animals have developed special chemical adaptations to increase the acuity of their vision or their sensitivity to light. Examples of such adaptations are intra-ocular colour filters and intra-ocular mirrors. Apart from the black and brown pigments of the eye which act as general light absorbers,

some animals have yellow substances in various parts of their eyes which absorb the scattered blue light and let through the yellow-green rays, to which the retina is most sensitive. Yellow filters are often used in photography to increase definition by absorbing scattered light which gives rise to haze, and also they are used to increase contrast. They do this by absorbing the same light-waves from different colour mixtures, thereby increasing the contrast between the remaining colours. In the last war, camouflage trees and grass were painted on buildings with paints that, although they looked green to the eye, were in fact made up differently from real grass green. If a yellow colour filter was used in observing or photographing these paints it showed up a contrast between the camouflage paint and the grass which had been previously undetectable. Yellow filters (spectacles) are also used by people on the look-out for planes, as they reduce dazzle due to scattered short-wave light. The yellow substances in the eye which have been evolved by different groups of animals presumably serve the same purpose as the extra-ocular filters of the photographer and the airman.

There are various kinds of yellow substances which act as filters in the eye, but one generalization can be made about them, and that is that they occur only in the eyes of animals that are active by daylight. Nocturnal animals that are exposed only to dim lights do not possess them. The human eye has the yellow carotenoid pigment, xanthophyll, just on the macula, which is the point on the retina where we have most accurate vision, and the lens, which is colourless at first, gradually turns yellow with age, so that whereas the lens of a child lets through 90 per cent. of the blue

light, the lens of a 78-year-old man usually lets through only 15 per cent—so do not argue about blue colours with your children! Some animals, lampreys, squirrels and prairie dogs, have yellow lenses throughout life. Some fish have bright yellow corneas, and other animals keep their filters in the light-sensitive cells themselves. The cone cells of the retinas of birds, frogs, turtles, and the primitive lizard, *Sphenodon,* contain yellow and red droplets which filter the light as it actually strikes the cell. In the chicken these oil droplets consist of substances chemically like the yellow pigment in carrots. The red oil droplets cut off even more light than the yellow ones, and in the turtles they are thought to cut out the intense glare off sunlit water.

At the back of the light-sensitive cells of the retina lies a single layer of cells, the pigment epithelium (p. 31). In man the size and shape of the cells and the position of the pigment within the cells is the same under all conditions of light and dark but in fishes and frogs when the light is bright the pigment migrates outwards between the rods and cones and acts as a shield, cutting down the amount of light that reaches the light-sensitive cells. On the other hand, in nocturnal animals who live under conditions where too little light rather than too much is the usual state, these same pigment epithelial cells, or an area of the choroid, may become chemically modified to act as a mirror, the tapetum lucidum. All light that falls on the retina is not absorbed, some passes through to the pigment epithelial cells and the choroid and if these are modified to form a mirror, the light is then reflected back again through the light-sensitive cells. This second passage of the light with the second possi-

bility that some will be absorbed by the visual cells can act as a second stimulus to vision; hence provision of a mirror will increase the stimulus given by light. This will be advantageous when the light intensity is low. Mirrors backing the retina are just the same in principle as the backing of the light-sensitive emulsion often found on a photographic film or plate.

The eye shine of animals at night is due to these mirrors. When you are out at night driving in a car or having a torch in your hand you can often see the glowing eyes of a dog or cat reflecting the light of your headlights or torch back to you. It is sometimes said that the human eye can flash but we have no mirror and in order to see the orange glow of the human retina, rather special circumstances are necessary. The light must be between the observer and the person and the latter must be long sighted. A few rare humans are born with a reflecting layer on the surface of the retina and their eyes often appear to have a golden glow.

Different animals have achieved a mirror in different chemical ways. Cats, dogs, foxes, badgers, and seals have a mirror-area in the centre of the retina which reflects a brilliant blue-green light. The reflecting surface is formed by myriads of crystals of a compound of the metal, zinc, with the amino acid, cysteine. So great is the concentration of these crystals within the cells that sixteen per cent. of their dry weight is zinc—their eyes could almost be called zinc-plated.

A golden mirror is found in the eyes of lemurs, made from crystals of riboflavin, the vitamin B_2 whose function is normally that of an oxidation-reduction catalyst in the body cells (p. 100). This is not only a

very beautiful and surprising mirror—the opened eye after removal of the retina glows like a sheet of burnished gold—but also acts as a kind of Day-Glow-Paint because riboflavin is fluorescent. When ultraviolet or very deep blue light falls on riboflavin it transforms it to a green light and glows green. The visual cells of the retina are not stimulated by ultraviolet light but respond maximally to green light so that a mirror that not only reflects visible light but also transforms ultraviolet light to green visible light is surely the apex of chemical adaptation to serve specific biological function.

The American opossum, *Didelphis virginiana,* which is a marsupial, has a white mirror made of crystals or globules of cholesterol and cholesterol esters. No other marsupial is known to have developed a mirror.

Herbivores such as cows and horses have also constructed a reflecting area in the choroid but do not use crystals. The mirror is formed from tiny regularly arranged collagen fibres which act as a diffraction grating and reflect a blue-green light.

Fishes and reptiles have yet another kind of chemical mirror, made of crystalline guanine. When Abelsdorf wanted to find out whether there was any change in the retina when it was illuminated he chose to examine the eye of an alligator as the retina of this animal is backed by a white shiny layer of guanine which makes any change of colour in the eyeground easily visible to an observer with an ophthalmoscope. He found that, before illumination, the retina was rose pink and that when light fell on it the colour faded. This mirror of guanine in the alligator's eye thus enabled the first observation of a light-sensitive pigment to be made.

CHAPTER IX

Cone Cells and Colour Vision

Light can cause a sensation only if it is absorbed by one or more photosensitive pigments in the retina. The cone cells of the retina do not contain detectable amounts of visual purple, yet animals that have nothing but cones in their retinas can see excellently in daylight, and one is forced to conclude that there must be some substances or mechanisms, other than visual purple, to account for the light-sensitivity of the cones. In dim lights, colours are not appreciated, which makes it reasonable to suppose that only one photosensitive pigment is operative, but in bright lights, colours can be discriminated and the colour matching properties of the human eye demand that at least three classes of photosensitive pigments be present. We can match any colour with a mixture of the three colours red, green and violet. As the brightness of the light changes, so does the relative visibility of coloured things. For example, in the dim light of dawn, flowers do not show their various hues; red ones seem black and others grey, but as the sun gets up, yellow flowers stand out against the grass, which still looks grey. The rod cells in the first light give colourless impressions, but as the light increases the cone cells first respond to yellow before there is sufficient light for them to react to blue, green or red.

We know quite a lot about *what* we see, but *how* we see it in colours—that is another matter. What is colour? As we have seen, red flowers may look black

and green grass grey, so that we can hardly suppose that the colour of a pot of geraniums is a property belonging to the flowers and leaves; it varies so much according to the light that strikes them. Is colour, then, a property of light? When Newton showed that a beam of colourless light could be split up into all the colours of the rainbow when it was passed through a prism it looked as if colour must be a property of the light itself. But since that time physicists have shown that the luminous spectrum is only a small part of the whole spectrum of radiation that is known to reach from the long radio waves on the one hand through infra-red to the visible light waves and so to the shorter waves of the ultraviolet and the X-rays. Most of these radiations are invisible to us, yet they are of the same character as that small band that gives us all our visual sensations, coloured as well as colourless. Our eye is insensitive to infra-red rays, which reach the retina, but have a wavelength which is slightly longer than the wavelength of the radiation which we appreciate as red light. A photographic plate is more sensitive to the ultraviolet waves, which do not reach our retinas, than it is to violet or red light. The effect of all radiation depends on the nature of the receiving organ. It is we who attribute colour to the pot of geraniums when our retinas are stimulated by the appropriate light waves. Colour, like beauty, exists only in the mind of the beholder.

Bearing this in mind, let us see what we know of colour from our own sensations and from the physical nature of light. The hue sensation depends primarily on the wavelength of light; the longest visible wavelengths arouse the sensation of red and the shortest ones give us the sensation of violet, wavelengths of

intermediate length producing sensations corresponding to the other colours of the spectrum. When all visible wavelengths are received together we see white light, but removal of any group of waves by absorption or reflection will make the remainder of the light appear coloured. If all wavelengths are absorbed, then the object will appear black. A normal person can distinguish about 160 colour shades in sunlight, yet it has been found that all these shades can be made up of varying mixtures of the three primary colours, red, green, and violet.

Newton started this investigation by taking small bits of the spectrum, mixing them and observing the result. Pure colours can be mixed by allowing parts of the spectrum to pass through narrow slits on a white reflecting surface. White light and all other colours (except brown and sage green) can be produced by mixing red, green and violet spectral lights in suitable proportions. This means that in order to see 160 different hues there is no need for the eye to have 160 different types of receiving cell and chemical substance, but only three different types, one for each primary colour. On this basis it has been suggested that the retina contains three different types of cone cells, one sensitive only to red, one only to green and one only to violet light. No one has found three types of cones in any one retina, and a single colour can appear as bright as white light, which is difficult to explain if it were able to stimulate only one-third of all the cones.

The electric response of the normal human eye when stimulated by a flash of light has recently been found to vary according to the colour of the flash. One type of electric response is evoked by blue and another

type by red light. These electrical changes were studied by putting one electrode on the conjunctiva and the other on the cheek so that the response to light could be measured without interference with the normal eye. The response to blue light was greatly increased by dark adaptation and was absent or small in the light-adapted eye. If the flash of light was restricted to the central area of the retina the response to blue light was much smaller than it was to a flash restricted to the periphery of the retina. In contrast to this the electrical response to a flash of red light was more prominent in bright light, was not affected by dark adaptation and was mainly derived from the central area of the retina. These electric responses appear to correspond in a general way, with the responses one would expect from the rods (blue light stimulation) and from the cones (red light stimulation).

Something more about colour vision can be deduced from our own experience of the way in which we can fatigue our light-sensitive cells. Apart from the three primary colours the spectrum contains pairs of colours which when mixed together give the sensation of white light; such pairs of complementary colours, as they are called, are red and greenish-blue, green and purple, yellow-green and violet. Now, you are well aware that after looking at a white light for a moment or two there remains in your eye when you look away, a black spot, just the size of the previous image of the light. This black spot moves with every movement of your eyes across the scene you are now observing, and then gradually fades away. If, however, you look at a bright red light you will get a greeny-blue patch in your eye when you look elsewhere, and

a green light will give you a purple patch for a short time. These are the pairs of complementary colours whose mixture forms white light. The theory is that when you gaze at a white light, you gradually exhaust the ability of your cones to be stimulated by light of any wavelength, and so the after-image is black, but when, for example, you exhaust your cones with green light they are still capable of responding to white-minus-green light, which is purple, and so you get a purple after-image. But if you look at some coloured thing and then shut your eyes, so that no more light gets on to your retina, you may see an identically coloured image of the thing you have just looked at. These positive after-images are due to the hang-over of the stimulus in your retinal cells which goes on acting momentarily if there is no other stimulus superseding it. These are the simplest forms of after-images, but you may get others, a series of differently coloured images for example—if the original light stimulus was intense and prolonged. Just as visual purple is bleached by bright light, so the cone receivers get fatigued and need momentary rest to restore their sensitivity to the kind of light that has caused their fatigue.

Colour vision may depend on the nerve connections of the cones as well as on the cones themselves. The 'wiring diagram' showing connections between the cones, the optic nerve fibres and the brain is complicated enough to support this idea. It has been worked out by the microscopical anatomists, employing the fact that nerve cells and fibres under certain conditions will stain black when they are immersed in a solution of silver salt, thereby, as it were, taking their own photograph. If thin slices, or sections, of the

retina are treated with silver and then examined under a microscope the nerve cells and their connections stand out sharp black against a grey-brown background and the picture looks for all the world like a wiring diagram for some Heath Robinson piece of apparatus. There are many different types of connection; even the position on the long cone cell at which the connection is made varies and may be of significance, and this complexity of connection may be the clue to the transmission of messages denoting different colours, from a cell which reacts to light of many different wavelengths (see Plate VI).

Not all people see colours combined from the three primary colours, red, green and violet. Colour-blindness is rather a drastic term for a defect of colour vision that affects about 4 per cent. of men and 0.4 per cent. of women in England and up to 12% of men in some parts of Australia. It rarely affects women, but they can pass it on to their children. It is congenital and incurable. Nearly all the so-called colour-blind see quite a range of colours, and if it were not for the fact that the colours they mostly confuse are reds and greens, they would not be so rigorously excluded from certain jobs. But as we use red and green lights for certain types of signalling, it is imperative that any man or woman engaged on work like this should have normal vision. John Dalton, the chemist who formulated the Atomic Theory of Matter, at the end of the eighteenth century, was the first colour-blind person to cause a scientific stir in England, though the condition was known much earlier. Dalton indeed said that blood looked bottle-green to him and a laurel leaf was a good match for sealing wax. His vision was much investigated by the physiologists, and

the name Daltonism was given to the red-green defect.

When a large number of colour-blind (or colour-defective) people are tested they fall into two groups, both of which match reds with greens, but the matches are quiet different. The smaller group matches a slightly blue-red with a dark green and the larger group matches a pink with a pale green. Many tests of a large number of severe cases of colour-blindness have shown that both groups will match every spectral colour with mixtures of only two colours, whereas the normal person needs three colours to get such matches. The colour-blind are therefore called dichromats and the normals trichromats. Their colour-blindness can be explained on the hypothesis that one of the three light-sensitive pathways in the retina and brain is missing. In the smaller group the 'red' pathway is missing, and they are spoken of as 'red-blind', or protanopes. In the larger group it is the 'green' pathway that is missing, and these people are called 'green-blind' or deuteranopes. Apart from these two groups there are many people who see red and green slightly differently from the normal, and when tested with spectral lights need either more red or more green in matching a spectral yellow. These are called anomalous trichromats; they cannot be called truly colour-blind but they are more dependent than is normal on the intensity of light and on the size of the object when judging any colour.

People have to pass a colour-vision test if they wish to work on railways, ships or aeroplanes, or as electricians. A common test is that of a specially constructed lantern which shows small spots of light of different colours, different sizes and different degrees of clearness. The person tested has to name the colours

correctly. Among the colours shown are signal red and signal green, and they can be varied to show as they would appear on a rainy night or through a fog. If the person tested muddles the colours when they appear in this way he is obviously not safe to be let loose on a job where red and green lights are used for signalling and for identification.

Another test consists of a series of white cards on which dots of different shades of red and green are arranged in patterns so that to the normal person a certain number—say 8—is apparent as a red figure on the background of green dots, but to the red-green colour-blind this number will appear as 3, since the missing part of the 8 is made up of red dots which are very slightly different in colour from the rest, which he will confuse with the dots of the green background pattern. There is a series of these ingeniously arranged so that in some the figure is apparent to normals and in some it is apparent only to colour-blinds. A very few people see no blue in the spectrum, but this anomaly is not often detected.

Just as the pigment, visual purple, absorbs light that falls on the rod cells and by catching the light-energy triggers the passage of a nerve impulse to the brain, so there must be a substance or substances in cone cells that absorb light of different colours. To absorb light the substances must be pigments, yet little success has been achieved in attempts to separate pigments from cone cells. In daylight the human eye is most sensitive to a yellow-green light of wavelength 560$\mu\mu$; one can therefore suppose that the major pigments in the cone cells will be most sensitive to light of wavelength greater than 500$\mu\mu$ though some mechanism for perception of blue light of wavelength

below 500$\mu\mu$ must also be present in the retinal cells.

Only two light sensitive pigments have been extracted from the cones of any animal. Wald was able to extract a mixture of visual purple and another pigment from the retinas of domestic fowls. These retinas contain predominantly cone cells. The extra pigment other than visual purple was also bleached by light and was most sensitive to yellow-green light of wavelength 560$\mu\mu$. This means that the substance itself must be a violet colour and Wald named it iodopsin. Iodopsin is a protein-retinene compound of the same general nature as rhodopsin. The protein of iodopsin is thought to be different from that of rhodopsin but the retinene is the same twisted form. The cone cells of the bird retina contain coloured oil droplets which can act as colour filters for the light falling on iodopsin. This arrangement would enable a single visual pigment to respond differently according to the colour of the light.

Recently Weale, in London, has extracted a very small amount of a light-sensitive pigment from the all-cone retina of the grey squirrel. Since this retina is made up entirely of cone cells, any pigment extracted must have been contained in them. It is therefore surprising and rather puzzling that the extracted pigment seems to be a typical visual purple in that it absorbs green light of wavelength 502$\mu\mu$ most strongly. Yet the squirrel's eye is most sensitive to a green light of wavelength 530$\mu\mu$. Bleaching of the solutions of the pigment extracted from the squirrel retina gives rise to another pigment with maximum light absorption at wavelength 480$\mu\mu$ (blue) and Weale suggests that the visual stimulus depends upon the proportions of these two pigments and that this would explain the fact that

the maximum sensitivity of the eye is different from the absorption maximum of the extracted pigment.

It is interesting that this advance in knowledge of the light-sensitive pigments of cone cells depends upon knowledge of the comparative anatomy of the retinas of animals. Squirrels are far from ideal laboratory animals and hens are macabre and unaccountable but study of their eyes may help us to understand our own.

No light sensitive pigment has been separated from the cone cells of the human fovea but Rushton in Cambridge has been able to detect two pigments with his ophthalmoscopic method which analyses the light absorbed by the retina under different conditions.

The relation of cone pigment to cone vision can most easily be seen in colour-blind people, particularly in the protanopes who are unable to distinguish red from green. Such colour-blind people are most sensitive to green light of wavelengths about 540$\mu\mu$. Rushton found that the pigment in the foveal cones of protanopes was bleached most readily by light of this same wavelength. As it is sensitive to green light it has been named chlorolabe. He could detect only this one pigment in the cones of protanopes and it seems probable therefore that this kind of colour blindness is due to lack of one of the normal light-sensitive pigments of the cones. No blue sensitive pigment has been detected but in the normal human fovea Rushton found two light-sensitive pigments. One of these is the same as the single pigment in the protanope, the other is sensitive to yellow-red light of 590$\mu\mu$ and has been called erythrolabe. The deuteranopes—or green-blind people—seem to have both pigments in their cones but do not use them in the normal way. Both chlorolabe

and erythrolabe regenerate after being bleached much faster than visual purple.

The question whether all animals that have cone cells in their retinas see the same variety of colours as we do or are partly or completely colour-blind, has provoked an immense volume of research, a large amount of which is inconclusive. The difficulty of decision is tremendous, owing to the fact that no animal can reply to a direct question, and that in testing preference for colours, or power to distinguish different colours, those used must have identical brightness, and the proportion of white in their make-up must be equal. If these conditions are not observed the animal may be able—as the colour-blind person does—to distinguish red from green not by colour but by brightness or whiteness. All our experience leads us to believe that birds and monkeys see colours. Think of the magpie and his passion for hoarding bright trinkets, or the brilliant colouring of male birds, which Darwin supposed to have survival value by being attractive to the female.

With other classes of animals the detection of colour vision is very difficult. In general, the problem has to be tackled by training an animal to associate food with a certain colour, showing it at the same time another colour with no food attached. When the animal makes the correct choice more often than not, the colour with no food attached must be gradually changed in intensity to see if really it is not colour, but brightness, that the animal goes by. If, at a certain change in brightness, the animal's training gives way and its expects food equally on either colour we can say that it is colour-blind. But if the training holds and the animal always goes to the same colour to get

food, however much the intensity of the no-food colour is changed, then we can say that it is truly able to distinguish that particular pair of colours from one another. You must then retrain your animal for another pair of colours. This is a long business. The tests must also be arranged so that smell, noise, position of food, presence of other lights, etc., cannot influence the choice of the animal.

As an illustration of a relatively simple experiment, let us take the investigation of colour vision in bees. Small squares of grey paper of differing shades but equal brightness were arranged like the squares of a draught-board, and one blue square was put among them. A very small dish for food was then put on each square and syrup was dropped into the dish on the blue paper. A bee was then let loose to see whether it could be trained to fly direct to the syrup on the blue paper. After a good many trials the bee recognised the blue-equals-syrup relationship, and then the pattern of grey squares and the position of the blue square were altered in each successive trial, but it was found that however the greys were varied the bee always chose the blue paper. If red was put down instead of blue the bee could not be trained to choose it. Red and grey of equal brightness appeared the same to it, and it had no idea where to expect the syrup. The bee was therefore considered to be able to see blue but to be insensitive to red. There is also some evidence that it can see further into the violet and ultraviolet than we can.

In spite of the work involved, the colour vision of animals has been intensely investigated. Fishes have been fed on red-coloured larvae and then deceived with red wool. Lizards which hate salt have been train-

ed to reject salt-soaked mealworms on coloured paper. Turtles, frogs, dogs, cats, rats, mice, hens and monkeys have all been studied. The case of the domestic hen will illustrate an interesting pitfall in such work. Grain was put down for the hens and lit up by a spectrum of coloured lights. The hens ate all the grain illuminated by the red, yellow and green light, but did not take the blue grain. It was therefore concluded that hens were blue-blind until someone found that it was possible to train hens to eat blue grain and that the only reason why they were originally shy of it was that it was not a normal occurrence for them to eat blue food of any kind.

To summarize the results of all this work, we can say no one has been able to prove whether fishes can see colours or not. Frogs are totally colour-blind; no one knows about the chamelon, in spite of his reputed ability to do a quick-change colour act. Turtles certainly distinguish colours, and birds can probably differentiate more hues than we can, particularly those at the red end of the spectrum. Finally, among the mammals, there is no evidence for colour vision except among the apes, monkeys and men.

CHAPTER X

Things that may go Wrong with our Bodies

The authors once asked a reasonably well-educated group of people what they thought were the causes of blindness, and how many possible ways of being blind there were. Most of them produced three, namely, to be born blind, to be blinded in war, and to get cataract. Asked what cataract was, the unanimous opinion was that it was a skin that grew over the eye. To the further question 'What is the difference between an optician and an oculist?' no one produced any answer at all. This book is largely the result of that depressing conversation. There are between two and three hundred reasons for impaired sight; cataract is loss of transparency of the lens, and an oculist is a qualified doctor, who has specialized in eye diseases and in testing for spectacles, while an optician is a qualified physicist who has specialized in the properties of lenses and spectacles and their application to various kinds of eye. The oculist could not make a pair of spectacles and the optician could not operate on a cataract. They can both prescribe glasses, and this is why people get them mixed up.

We obviously cannot go deeply into the question of eye diseases, since, even given a preliminary medical background of six years during which the structure and diseases of the whole body must first be learnt, their study entails the digestion of about five thousand pages of print and roughly three years' study. We can, however, from our knowledge of the way the eye

works, think out for ourselves some of the ways in which it can go wrong. Before doing this, however, we must consider broadly what kinds of things do go wrong with living bodies and their organs.

First of all something may go wrong with the shape, size or structure of a part before birth, so that the baby may be born with a part missing, a part in excess (though this is rare), or a part misshapen. For example, some people have six fingers, others have only three, at birth. The extra finger is usually cut off, since we value conformity so highly, but the missing finger in the other case cannot be supplied. Similarly a baby may be born with one eye, or both, missing, and nothing can be done about it, since even if an eye could be grafted in, the connections could not be joined up with the brain and no message could reach consciousness. Such defects of structure are said to be 'congenital'. There are many minor ones, which do not matter, and some which, though their seeds are present at birth, may not be apparent till later. An easy example of this is seen in those people who have one blue eye and one brown. Both eyes were dark blue at birth (as are all European babies' eyes) and during growth one became light blue and the other brown, because the structure necessary for odd eye colour was present in the fertilized egg cell from the beginning. Such anomalies may by the purist be called 'developmental', as distinct from congenital, which means literally present at birth, but both groups are the same, as they are both disorders of growth. Sometimes these congenital anomalies are quite unpredictable, but there are a large number which are hereditary and the possibility of which can be foreseen. Sometimes they are directly inherited

from father to son, sometimes they skip a generation, and often they are passed on through a normal mother to all her boys, her daughters being apparently normal, but capable of transmitting the defect to their sons in turn. This form of transmission is said to be 'sex-linked'. The most widely known example of it is haemophilia, the haemorrhagic disease which has been known for centuries to affect certain of the males of the Russian, Spanish, English and German royal families; its descent has been traced to a certain German princeling who appeared in the Hapsburg family very many generations ago. Its inheritance is completely understood, and planned marriages could bring it to an end at once in these and other similarly affected families.

There are very many minor deviations from the normal which are developmental and which we do not consider as diseases. Indeed, all the minor differences between individuals are of this nature. If we all grew and developed alike we should be indistinguishable from one another (as ants and flies and frogs are to us, though probably not to their own kind), but as it is, one person has a longer nose, another a thinner lip, another has the right eyebrow a fraction higher than the left, and so on. These minor differences are, of course, accepted quite calmly until they involve some structure as sensitive as the eye or ear. Then we begin to wonder if it is all right that Mary cannot read the number on the bus quite as far away as we can, or that John gets confused over colours, but can count more barrage balloons on the horizon than we, and that Tom cannot hear a bat's squeak, but does not seem to be deaf. It is not surprising that these differences exist in healthy people, and when we realize

that an alteration of a millimetre in the distance from the back of the lens to the macula will make all the difference between long and short sight, the wonder is not that people vary, but that we all do turn out as much alike as we do.

The next group of diseases are those due to injury. There is no need to labour these: a broken leg, a bruise, an eyelid scarred from a burn, a wrist stiff from a fracture, a limb numb or paralysed from a severed nerve, all these are due to injury and are called 'traumatic', which means the same thing.

Next we come to the inflammations and infections. These are diseases due to the invasion of the whole body, or of a part of it, by micro-organisms or germs, as they are still popularly called. Some of these little creatures are extremely small and are called 'viruses'. These cause infectious diseases, such as measles, small-pox, chicken pox, shingles, and many others, most of which are generalized illnesses with fever. They may involve the eye in common with the rest of the body, and sometimes the eye alone.

Other micro-organisms which are larger and are called by various names descriptive of their shape (bacilli, cocci, spirillae, mycetes, etc.) may be lumped together as microbes. Many of these are extremely beneficial to man. Without them we should have no crops, no cheese, no vinegar, no beer. A number, however, are extremely harmful, and when they invade the body (infection)—through a scratch, an insect bite, or by being breathed in or swallowed—something has to be done to fight them. The reaction of the body to these invaders is called inflammation and takes various forms according to the bacteria, but in most cases redness can be seen because more blood

must be brought to the part (which therefore swells and becomes tense, hot and painful), and finally pus is formed in bad infections. Pus consists of the dead bodies of those white cells of the blood, the soldiers and policemen of the body, which have died repelling the invader. Inflammation is like a battle. If the defensive power of the body is strong only a little redness may occur and pass off. If it is not so strong, or the enemy is stronger, then pus will be formed before the wound heals. If the invader is too strong for the body, then a generalized infection occurs which may end in the victory of the bacteria and the death of the man. The scientific side of medical treatment aims at assisting the defensive mechanisms of the patient in various ways, i.e., killing the invaders with chemicals, bucking up the white cells, helping the body to produce chemical defences itself, letting out the pus, cleaning and disinfecting the site of invasion, and so on.

Inflammations vary in intensity. They may be acute, sudden in onset, and severe. They may be chronic, running a course not too severe but never quite clearing up, partially disabling without killing.

Next we come to another kind of disease, the tumours or new growths. These are difficult to define and are as yet not thoroughly understood. Tumour means a swelling, but it is used by doctors to mean only those swellings which are due to overgrowth of tissues and not to inflammation. Some growths, for example fatty tumours, are quite harmless, and annoying only because of their appearance, since no tumours are of themselves painful (they give rise to pain only when they press on surrounding parts). Other tumours are malignant, and this type is charac-

terized by unchecked power of growth and by the breaking off of little bits of the original tumour, which are then carried by the circulation of the blood and lymph to other parts of the body where they start to grow and produce other tumours, like the first. The cancers belong to this class of malignant growths, and there are others.

We have so far taken no account of a group of diseases which become more common with age. These are called the degenerations. The greying of our hair, the hardening of our blood-vessels, the wrinking of our skins and the loss of elasticity of our lenses are examples of these. Some of these conditions are due not to age, but to malnutrition, such as the degeneration of the rods in the retina when they are deprived of vitamin A. Somewhat allied to these degenerations are disorders of individual organs, for example the kidneys, the heart, the thyroid and pancreatic glands. Some diseases of organs are the result of previous infections, others have obscure and as yet unknown causes. Their symptoms will depend on the organ involved. For example, if certain cells in the pancreas go out of action the patient cannot make use of sugars and starches in the food, so that they pass out in the urine and the patient has diabetes. This condition of things will, if untreated, cause a variety of side effects (due among other things to the circulation of sugar in the blood), some of which will affect the eye. The healthy functioning of the retina and lens will be interfered with, and by examining the retina with an ophthalmoscope the oculist can see that the patient has diabetes, often long before he is aware of anything being wrong. A number of diseases of this nature produce alterations in appearance of the inside

of the eye and can be discovered by a medical eye examination. Kidney disease, brain tumour, some forms of anaemia, and high blood pressure can all be discovered in this way. This is one of the reasons why it is better to go to an oculist than an optician, who may not notice these signs of other diseases.

Then, in addition to all these things which may go wrong with us, there are the disorders of function which are in a sense diseases which we produce ourselves. For example, many a student gets diarrhoea before examinations, but this does not mean that he has an infection (such as dysentery, which produces the same symptoms). If the student realizes that the symptom is caused by his anxiety he will find that it subsides and all is well. But if, as sometimes happens, he does not recognize its mental origin he may become seriously alarmed about himself, which will increase the symptom, which will in turn alarm him more, and so on, so that a vicious circle will arise, only breakable by realization of the original cause, which was anxiety, and arose in the patient's mind. This type of illness, mental and emotional in origin, often obscure to the patient, but producing physical effects, is very common. Fear plays a large part in its production, and we can easily see how fear can lead to a vicious circle of symptoms. The physical manifestations of fear are many; palpitation of the heart, rapid breathing, prickling of the skin, nausea, diarrhoea and vomiting, faintness, restlessness, sweating, and many others, according to the intensity of the fear and the type of person experiencing it. If the origin of the fear is a very obvious one—a mad bull, an earthquake, an air raid—we have no difficulty in recognizing our fear for what it is, and it will then disappear with the

danger which caused it, but if the origin of the fear is something more subtle than this, a private worry or anxiety, a sense of insecurity or an apprehension having no foundation in fact, the victim may experience the symptoms without relating them to the cause, and so may start a vicious circle of fear —> symptoms —> fear of the symptoms —> more and more symptoms —> more fear, until he has become really ill and has lost all memory of what started the business. Sooner or later the symptoms will be related to one or other part of the body almost exclusively. This may be because that organ is congenitally weaker than the rest of the organs, and so more easily goes wrong, as when a person with a weak digestion develops the symptoms of a gastric ulcer under anxiety, but quite often the organ affected is determined by the nature of the fear and is itself quite healthy, as when an artist develops severe headache and eyestrain through fear that he will be unable to work if he loses his sight. This sometimes acts the other way round, to get the person out of a job he does not like. Thus, I earn my living by adding up figures—I hate it—it gives me a headache—I get a headache after adding up figures—I expect it hurts my eyes—I ought to have glasses—I see an optician's advertisement about eyestrain and get his glasses—this figure job has ruined my eyes—I never had to wear glasses before—I ought to give it up—I am quite justified in getting ill and giving it up as I have bad eyes—I must not use my eyes—must not read at all or I shall get a headache—and so on and so on, till the original state of mind is quite forgotten and the person has become a victim of his own emotions, which have got him out of the job he hated, but have made it impossible for him to enjoy

any other. Such people are in no sense malingerers. They are not pretending, but they lack insight into the working of their own minds and above all they lack confidence. They can be cured by anyone, orthodox doctor or unorthodox 'nature-curer' who will take the trouble to restore their confidence and remove their fear, but this requires immense patience and understanding, which is rare!

We can now begin to apply our knowledge of the kinds of diseases (pathology) to our knowledge of the structure of the eye (anatomy) and make a shrewd guess at the nature and causes of poor sight. We will work on an anatomical basis and consider first how things can go grossly wrong with the structure and mechanism, then how size and shape without disease can alter sight, and then how function can be impaired by wrong usage and misunderstanding.

CHAPTER XI

Things that may go Wrong with the Mechanism of Seeing

This chapter tries to show extremely briefly how the mechanics of seeing may go wrong from various causes. A discussion of the causes would be far too difficult, since a general medical training is necessary to appreciate many of them, but we will try to show how a doctor discovers what is wrong by thoroughly understanding the mechanism and then working out where it must have broken down to produce the effect the patient complains of. He does this in much the same way as an expert motor mechanic, to whom an ignorant owner-driver has complained of engine overheating. He knows that the owner may have forgotten to fill the radiator, or to remove the muff, that the fan may have stuck, that the fan belt may have broken, that the radiator may leak, that there may be a block in the circulation of the water, and so on. He then proceeds to examine the car carefully to find out which it is. Often it is something quite simple and he sees it in a minute, but sometimes he may have to run the car himself and then take it partly to pieces before he can find out just where it has gone wrong.

Similarly, an oculist may be able to glance at a patient to know he has an ulcer on his cornea or an inflammation of his iris, or he may have to do a long examination, mapping out the fields of vision, examining the function of the retina in bright and dull lights,

testing the acuity of the eyes separately and together, getting the patient's blood pressure and urine tested and analysed, and finally having a complete overhaul of the whole body, before he can decide just where the breakdown really began. In the end he can always find out why the patient cannot see properly, and he is very likely to be able to cure him, but what he usually finds more difficult than anything else is to explain to the patient what is wrong. This is because the average person has so little knowledge of how his body works that he and the oculist, who has studied it all for at least ten years, have no language in common. The patient, unless he is quite young and has been taught a little physiology at school, will have no conception of the basic facts of which you now have a little knowledge if you have read thus far. Where is the doctor to begin the explanation if the patient thinks his eye is a shiny blue or brown circle with a round black spot in the middle? The doctor is often accused of obscurantism and the nature-curer or faith healer is hailed as a prophet because he tries to unify all disease under one cause with one universal cure and is therefore comprehensible. This is really as silly as saying that automobiles always overheat when the owner has forgotten to renew his driving licence.

The mechanical side of the study of eye diseases is therefore largely a matter of common-sense application of certain principles to a thoroughly understood mechanism, and we will show briefly how this is done. True eye disease is, however, not so common as are variations in the shape, size and function of healthy eyes. These will be dealt with in a later chapter, since even more misapprehension exists about them in the public mind than about the diseases.

We now know that the rays of light from the thing *looked at* go through the cornea, through the aqueous, through the lens, through the vitreous, make a picture on the macula, set up an electric current which carries the picture through various brain centres to consciousness and we *see* the thing looked at.

What may go wrong? Much is obvious. The cornea may not be clear and smooth. Sometimes people are born with opaque corneas, but this is rare. Mostly the cause of opacity is an injury or an inflammation. Nutritional diseases (lack of certain vitamins), venereal diseases, and virus diseases often affect the cornea, and of course if it is not clear the formation of a proper picture on the retina is hindered from the very start. How badly the person sees depends on how dense the opacity is and in what position. If it is at the side it will not matter. If it covers the pupil area it will matter a good deal. If it is in the centre of the pupil it will matter more in bright lights, when the pupil is small, than in dim lights, when the pupil dilates and the person sees round the edge of the opacity. If the cornea has been cut (as by flying glass in air raids) it will heal up neatly but will often lose its regularity of curve, so that the person sees things distorted, as through a piece of wavy glass. It is obvious that for good sight the pupil area of the cornea must be clear and of the correct regular curve.

If sight is impaired by hindrance of the passage of the light rays at the surface of the eye it is difficult to know what to do. The position is, however, not hopeless. Often quite dense scars in the cornea clear remarkably in time, and even if the whole pupil area is opaque the surgeon can sometimes cut a little bit out of the iris opposite a clear part of the cornea and

the patient can see through this. It is also possible to graft a clear bit of cornea from another eye into a scar, and this is sometimes wonderfully successful, but usually these procedures do not produce full normal sight. They are, however, very much better than nothing, for to a person quite blind even a tenth of the normal seems miraculous. There is another way of dealing with corneal scars which are not very opaque but which are irregular and so produce distortion. This is by means of contact lenses. Most people have heard of these, but they consider them either as the film star's substitute for spectacles, or as a magical cure-all for bad sight. Neither conception is accurate. A contact lens is a thin shell of glass or plastic which fits over the cornea inside the lids, not quite touching it but separated from it by a thin layer of fluid (tears or salt solution). If you have understood the way eyes see under water you will get the idea, which is this. The glass, the saline, the patient's cornea and his aqueous are all about the same optical density, so that the only bending of the rays of light will take place where they leave the air and enter the glass. As the glass can be made to any desired curve, what we have done is to substitute a nice smooth regular glass cornea for a wavy irregular scarred one, which, being inside the glass, no longer affects the path of the rays. (A similar phenomenon occurs when we render ground glass transparent by wetting it and thus filling up the irregularities.) Sometimes the improvement in sight is almost miraculous, and this method is always worth trying when the defect is due to something wrong with the evenness or curve of the surface of the cornea.

It is very difficult to make contact lenses so that

they can be worn with comfort. They are not substitutes for spectacles, but are for a different set of diseases which cannot be much helped by spectacles.

When the light has safely passed through the cornea it has to cross the anterior chamber and enter the pupil. The aqueous may be muddy and thick (from inflammation), not clear like water, though this is a rare cause of poor sight. Again, the iris may be stuck to the lens from inflammation and the pupil may fail to contract and dilate and may even get stopped up with pus, etc., in inflammation, so that the light cannot get through.

Next we come to the lens. This should be clear and of the right curve and capable of altering its curve in accommodation. Two things may go wrong with the lens. The first is loss of elasticity, so that the curve becomes fixed, and the second is loss of transparency. The loss of elasticity of the lens is not strictly speaking a disease and does not have serious results. It will be dealt with in the next chapter. The loss of transparency of the lens is, however, another matter, since if it becomes complete it obviously prevents the person seeing anything at all. The opacity is, however, practically never complete, and so the patient can sense the difference between light and darkness. Some diffuse light will get through, just as it gets through our eyelids when our eyes are shut. Loss of transparency of the lens is cataract. It is a queer name, meaning to fall or drop down (hence a waterfall) and was first used by people who thought a sort of opaque shutter had dropped down behind the pupil and did not realize that it was the lens itself which had gone cloudy. It is a pity that we have only the one name for any kind of lens opacity, however small, since

most people connect the word cataract with blindness and operations, and so get frightened and fall a prey to all kinds of semi-magical treatments supposed to cure it. They do not usually even know what it is, and often think it is an opacity in the cornea or even a new layer of opaque skin which in some mysterious way grows over the whole eye. In actual fact small opaque spots in the lens are extremely common and do not interfere with sight at all, any more than a few specks of dust on a camera lens prevent us taking a picture. At least 25 per cent of normal people have some little imperfections in their lenses which are, technically, cataract, but about which they never know unless someone tells them and which never get worse. These imperfections get commoner with age and in a certain number of cases do increase until they begin to interfere with sight. But so long as even a little bit of the centre of the lens remains clear the person can see. Try an experiment about this. Take an ordinary magnifying glass and a piece of paper. Catch the picture of a window or an electric light on the paper with the lens (as we did in Chapter III) and then with a brush and some ink put a lot of spots and blots all over the surface of the glass. You will be surprised how much of the lens you have to black out before you can stop it forming a picture. The picture will get fainter, but will not disappear until you have covered nearly all the lens. This gives you some idea of how much 'cataract' a person may have and still be able to see quite happily, especially with a good light, and disposes of the popular sequence of ideas, cataract—blindness—horrible operation. Many people over seventy show a few spots of cataract, but in the majority the increase is so slow that the person

is killed in an air-raid or a motor smash or dies of old age before his sight is seriously interfered with. If this were more generally realized and if it were also known that oculists can often see signs of cataract ten or more years before the patient notices any deterioration of sight, people would not be so apprehensive and so anxious to find cures which will stop the progress of the opacity. From time to time many such cures, some silly, like staring at coloured lights, some rather irritating, like eye-drops of salt solution, some simply directed towards improving health, like vitamins, are vaunted and tried out both by doctors and quacks. One can always get heaps of 'cures' from any of them because most cataracts are so slow in developing and a few even spontaneously improve. No true medical cure for the ordinary slow cataract of old people is known or is likely to be known until we know the cause of the opacity, which is still a mystery.

There are some kinds of cataract of which we do know the cause. In some cases the opacity is hereditary and even congenital, in others it is due to diabetes or to kidney disease and to various other general illnesses, so that anyone with a slight cataract should be overhauled to see if there is anything else the matter. There may be a nutritional factor, and also certain occupations predispose to it. These are the trades in which the eyes are exposed to heat and infra-red rays, e.g., glass-blowing and smelting. These are now understood to be dangerous, so that proper protection for the eyes must be provided or cataract will in the end occur.

Let us suppose that a man has the bad luck to have a cataract which is progressing and getting more

dense. What happens? He finds that his sight gets more misty, that he is dazzled in bright sun and prefers to sit back to the light, that he requires a brighter light to read by and that he sees less and less well until finally he can only tell light from darkness and can just see the direction from which the light comes. (If he becomes unable to tell light from darkness there is something wrong with his retina as well, he has more than a cataract.) At this stage, or sooner, he goes to an oculist, who operates on his eye. The object of the operation is to remove the lens from the eye so that the light rays can get through the pupil again. There are many ways of doing this, some of them known in the East more than a thousand years ago, some of them of recent discovery.

The operation is not painful, nor does it take long, neither is it necessary to wait till all sight is lost before having it done. Let us think what has happened now to our patient after his operation. He has an eye with a cornea which is clear, a pupil, a vitreous and a retina, but no lens. The rays of light will be bent a little at his cornea, but not enough to form a picture on his retina, so that he will not really see much better at first. He is therefore given a spectacle lens of a curve equal in effect to that of his own lens, which he has lost, in order to bend the light sufficiently to make a picture. He can now see beautifully and is at first delighted. After a bit, however, his transports abate because of two things; firstly, he cannot accommodate and must have two pairs of spectacles or bifocal glasses to be able to see at different distances, and secondly, the effect of a spectacle lens is not the same as one's own lens, and objects look bigger and

flatter and nearer than normal. Add to this the fact that only one eye has been operated on and so he has lost his stereoscopic sight, and you have the basis for a host of complaints. He says he feels giddy and unsafe, for the world looks flat and unnatural and all on top of him. He trips over steps and drops his tea-cup from misjudging the distance of the saucer, and gets into a heap of minor difficulties which sometimes obscure the fact that he can see to read and write easily and is no longer blind. After a bit he re-learns his judgment of distance and manages to cope with the spectacles and finally admits that it was well worth it, and he is now quite comfortable. People vary in the time they take to adjust to all this. The younger ones are naturally quicker, but the operation is well worth while at any age. If the second eye is then operated on the stereoscopic sight returns completely and all is well.

If, therefore, one had to choose which potentially blinding eye disease one would rather have, the obvious choice is cataract, because, first, one may have it for years and never go blind at all, and, second, the operation will restore sight. A disease causing opacity of the cornea is much worse, since there is no certain way of curing this, and a disease of the retina is obviously worse still, since if the photographic film is spoilt the picture won't come out at all, however good the camera.

This brings us to diseases of the back part of the eye. Nothing much goes wrong with the vitreous jelly, except that sometimes the 'floaters' in it increase from various causes, so that they become a real nuisance and look like a lot of smuts all over everything. There is often something wrong with the person's health

when this happens, and this should be dealt with.

Next we come to the retina itself and the choroid which nourishes it. They may both go wrong together in a variety of ways, some of them obvious in their causation, as when, after a blow on the eye, the retina is torn and peels off from the pigment layer (detached retina), and some of them from quite unknown causes, Sometimes the changes which occur are hereditary and sometimes due to inflammation. Sometimes they are simply due to old age and very often they are really caused by general diseases—we must again mention diabetes and kidney disease among others. In any case, what happens as far as the patient is concerned is the same, namely, that where the picture falls on a damaged bit of the retina it is not registered properly and a bit of the surrounding scenery is blurred, distorted, or absent. Exactly how this affects the person depends of course on which bit and how much of the retina is affected. We can easily think this out. Supposing something goes wrong with the macula. This is the point of greatest sensitivity, and the most minute fault in it will be noticed at once. There will be a blur or distortion or disturbance just in the centre of everything looked at. The rest of the field of vision will be all right and the person will be able to walk about with ease, but will not be able to read or see anything with much detail. Such patients often ask why stronger glasses do not improve their sight, but it is obvious that however well the rays of light may be focused (which is all that glasses do), if the receptor cells are not working the picture will not be seen. Sometimes if a few cells are still all right the person can read with a magnifying glass because this spreads out the picture over more cones and there

is a greater chance of its falling on one or two good ones. This does no harm. Most people with chronic macular disease become unable to read, but do not go blind because our real independence of movement depends on having a big field of vision. They are in the position of rabbits, dogs, and many other mammals who have no macula anyway and get on all right, as they never want to read.

Suppose now that the macula is all right, but the rods (which see in dim lights) have gone wrong. Such a person will see colour and fine detail and will get around fairly well in bright daylight, but as soon as it gets dusk he will have lost most of his peripheral field and will start to bump into things. When it is darker (say starlight only) he will be practically blind, as his cones will then hardly function at all and his rods have gone wrong. Such a person is said to be 'night-blind'. There are many causes for this. One, as we have seen, is deficiency of vitamin A. There is another form which is hereditary, and there are others besides. We must not, however, mix up these people who have actual structural defects with the host of others who are simply afraid of the dark and do not try to see in a black-out. Many town-dwellers in England who were never used to unlighted lanes and dark woods were much more bothered by the 'black-out' of the big cities than were the country people who had always known what night was really like. These townsfolk would find that with practice they could learn to see as well as the country people. The truly night-blind person never improves with practice, but is continually in the state of those diurnal birds and animals who have only cones in their retinas and get up and go to bed with the sun.

Sometimes, owing to various causes, both rods and cones in the peripheral part of the retina go out of action and there is a blind area in the field of vision both in bright and in dull lights. This, unless it is so large as to leave the macula only functioning, is very often not noticed by the patient until he begins to find that he trips over things or that people come suddenly upon him without his noticing them. Some of these small losses of field mark the beginnings of serious disease, and it is important to find them out. They may pass unnoticed if the person simply buys a pair of glasses, but can easily be detected by a medical oculist. Among other things they mark the beginning of a very insidious disease (glaucoma) which in its early stages can be stopped, but cannot be cured if it is left too long and may lead to incurable blindness. (See Plate VIII.)

So far we have contrasted loss of central with loss of peripheral vision. There are several other ways in which the field can be affected which we can work out for ourselves. We can see the possibility of at least three. The first is a patchy loss, such as would be caused by a lot of small areas of the retina going wrong, say from broken blood-vessels or patches of inflammation; the second is a general dimming due to failure of conduction of the electric message along the optic nerve; and the third is a loss due to some block in the perceiving centres in the brain. (There are a variety of primary causes for alterations in the visual field which we need not go into here. All we are trying to do is to show how the machinery may break down, rather than the ultimate causes of the trouble.)

If there are several small blind areas in the field they are not usually noticed unless they are very near

Plate VIII. Eight different appearances of house and scenery as they appear to—

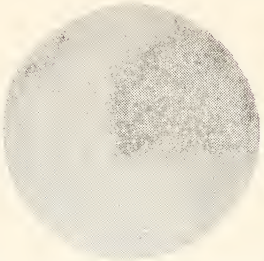
a. a normal person.

b. a totally blind person.

c. a person with a full field of vision but imperfect definition all over it.

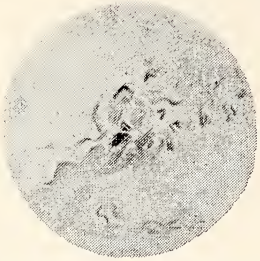
d. a person with a defect of the macula.

e. a person with complete cataract (perception of light only).



f. a person with some appreciation of the direction of light.

g. a person who has lost the peripheral part of the field of vision.



h. a person with a defective or detached retina.

the macula. Further out they will hardly bother one at all. Often they are discovered only in the course of an oculist's examination, when they may throw valuable light on the patient's general health, the state of his circulation, his blood-pressure, his nutrition, and many other things.

If the conductivity of the optic nerve is lowered the picture as perceived by the brain will be dimmed, sometimes all over, sometimes more in the centre, and the apparent brightness and hue of colours may be altered. This may occur from a number of causes, some of them poisons which people take themselves, such as 'hooch', the impure wood alcohol which blinds by upsetting the optic nerve, and the poison in shag and cigars which affects the sight of pipe smokers and millionaires after years of heavy smoking. Contrary to popular belief, this practically never happens to cigarette smokers unless they roll their own cigarettes from shag. Cigarettes may upset people in other ways, but they tend not to produce partial blindness as does pipe smoking. Anyway, this is unimportant, as if the person will give up his pipe or his cigar his sight will return all right. One merely has to choose between the pipe and the newspaper, as one can no longer have both. Most people choose the newspaper.

There are other causes of defective conduction in the optic nerve, from some of which recovery is possible. Some of them are related to disease of other parts of the nervous system and of the brain, and often by examining the end of the optic nerve, which is visible in the eye, the physician can tell much about what is going on inside the skull and elsewhere. Sometimes the patient's life is saved by a timely examination of the retina, for early signs of serious disease

show here before the patient notices anything wrong at all.

Supposing the eye and the nerve are all right and the picture is formed and telegraphed quite correctly, but the receiving station in the brain is out of order. If it is completely out of order the patient will, of course, be blind, although his eyes are perfect. Usually, however, one half of the receiving station only goes wrong, and then one half of the picture does not register. The effect of this is rather peculiar, and to understand it we must go back for a moment to a bit of anatomy. The picture from each eye is telegraphed along each optic nerve to the brain where there are various relay stations before it gets to consciousness. In the course of these relays the pictures from the two eyes get mixed up so that the right and left halves of the brain each receive a message from each eye. In some animals the whole picture from the right eye goes to the left side of the brain and *vice versa,* so that destruction of one side of the brain would lead to blindness in the opposite eye only. In us, however, the right half of what we see with either eye is 'seen' by the left half of the brain and the left half by the right side of the brain, so that destruction of one half of this visual part of the brain will not lead to blindness in one eye, but to loss of the same half of the field of vision in both eyes. Thus a man with a little bit of shrapnel in the visual part of the left side of his brain will not see anything to the right of the mid-line with either eye, and *vice versa.* Some readers will understand this quite well because they may have experienced it as a transitory condition with a certain kind of migraine, or sick headache. Sick headaches are very common, and some people who are subject

to them get a warning just before one is due. This sometimes takes the shape of a jazzy light which flickers, usually just to one side of where they are looking, and sometimes appears as a complete blotting out of the right or left half of the whole visual field. Hence, probably the term 'blinding headache'. This migraine blindness is never permanent. It is a mysterious disease, usually having nothing to do with the eyes at all, and much more research will have to be done before it is understood. The seat of the visual symptoms is in the brain, not in the eye.

To return to the person who has permanently lost the same half of his field of vision in each eye. If he has lost the right half and is a European or American he is very handicapped, as he can hardly read. This is because when reading from left to right we are constantly jumping on ahead to see what's coming, and this he cannot do as the right side of everything is blank. If he is an Arab he will not mind, as his writing goes from right to left and he can see what is coming, and all that disappears is what he has just read, anyway. Try this by moving a piece of paper across the page as you read. If you move it along behind the words as you read them it makes no difference, but if you pull it ahead of you you can read only very slowly. If, however, you turn the book upside down but still push the paper from right to left you will read more quickly, and some people who have lost the right half of the field of vision can train themselves to read fluently upside down.

Apart from these ways in which seeing can be interfered with, things can go wrong with the structures outside the actual eyeball. People often think that one involves the other. A parent will bring a

child to an eye hospital and say 'He has always had weak eyes since measles, doctor,' meaning that the edges of the eyelids have been inflamed, red, and crusted, but the child's sight may be perfect and the eyeball perfectly healthy. There is no necessary connection between sore lids and poor sight.

Sometimes the muscles that move the eyes go wrong. If one of them, say the muscle that pulls the right eye to the right, goes out of action, the person will not notice anything as long as he looks to the left, but as soon as he tries to look to the right the left eye will turn properly, but the right eye will get stuck and he will see double because his two maculae have not got the same picture on them. This is a kind of squint, though not the common kind, which we will deal with later, as that is a disorder of the way the eyes are used and not an actual failure of any one muscle to act.

CHAPTER XII

Things that may affect the Function of the Eyes, apart from Disease and Injury

We now come to the chapter many readers have been waiting for—about spectacles and headaches and eye exercises and squints.

First of all, what is meant by an error of refraction? This always seems a difficult thing for people to grasp. Some think it is a frightful disease which will blind them, others think it isn't there at all and they imagine it, or that they produce it by straining (what and how not stated), and others think it arises in some way by the interaction of mind and muscles and can be charmed away by yogic practices and psychology. Let us tackle it from the mechanical aspect first and see how these peculiar superstitions have grown up.

First of all, what is the normal eye? If you are a cod-fish it is normal to see objects near to you clearly unless you contract your little focusing muscle and pull your lens back towards your retina to get the picture of distant objects sharp. If you are a shark or a man it is normal to have a sharp picture of distant objects on your retina and to have to use your focusing muscles to see near objects. The ideal human eye is held to be one of such a size, shape and regularity of corneal and lens curves that distant objects are focused clearly without the individual using his ciliary focusing muscle at all. If he wants to see near

objects he must use his accommodation (contract his ciliary muscle). He must therefore have a good elastic lens to give him a good range of focusing; he must also have the muscles which move his eyes in his head all of the right length and equally balanced in pull one against the other, so that his two eyes look straight forward when at rest and converge (turn in) properly together when he accommodates for a near object. Such a paragon is a rarity. Only one or two in a hundred people between the ages of ten and forty-five can be allowed into this select group of the theoretically normal. This is not surprising when we remember that we are dealing with a living structure acting as an optical instrument and with a purely arbitrary definition of what should be considered 'normal' for it. We might easily have decided to take the 'majority' eye as our normal and so consider that a certain amount of long sight and astigmatism (see later) was desirable, since most people have it. It would be surprising, indeed, if every eye, or even nearly every eye, turned out to be alike. How few people resemble one another in feature, in height, in colouring, and yet the proportions of their eyes differ only by a sixteenth of an inch or so in most cases as it is. A sixteenth of an inch can, however, make a big difference to the properties of an optical instrument. What is really amazing is not that so few people have theoretically normal eyes, but that anyone does. The eye is actually one of the most uniform organs of the body, differing very much less from person to person than, say, length of leg or size and shape of jaw, but because the eye is an instrument of great precision we can measure and can ourselves be conscious of extremely minute deviations from our

ideal type. These small differences in the dimensions of the eye produce alterations in the optical properties called errors of refraction. 'Errors' is rather a strong term for them, as it is apt to imply something slightly reprehensible.

There are two common ways in which the dimensions of the eye vary: first in the distance from the centre of the cornea to the macula, which theoretically should be 0.945 inches, and is usually a fraction more or less than this, and secondly in the shape of the curved surface of the cornea, which 'should' be a portion of a sphere of radius of 0.30 inches. Both these dimensions, of course, are altering slightly all the time a baby is growing, because its eyes have to grow with it and are not so large at birth as they are in the teens. Growth continues to a slight extent until twenty-two or twenty-three in some people, but the kind of eye a person will have is roughly determined by fourteen in most people. The final shape of the eye and how it will work as an optical instrument is largely determined by heredity, 'long' and 'short' sight tending to run in families and to be as much a part of the individual's general make-up as the shape of his hands or the texture of his hair. It has never been proved that, apart from nutritional factors influencing growth (which, of course, may modify any bodily structure), the shape of the eye can be altered or influenced in any way. What can be profoundly influenced is the use we make of the kind of eyes we happen to have.

Let us think about the effect on the possessor of what is called long sight. Rather more than two-thirds of the inhabitants of Britain and America are long-sighted, and this majority is responsible for

certain of the patterns of our civilization, for example, our seating arrangements in cinemas and theatres, the size and position of public notices such as signposts, the use of the blackboard in schools and others of our common arrangements, which presuppose clear sight for things more than six yards away. Had it happened that the two-thirds majority had eyes of a diameter of more than 0.945 inches, all these things would have been different.

Now, what exactly is 'long sight', or hypermetropia, as it is called? Structurally it means that the distance from the front of the cornea to the macula is a little less than the rare so-called 'normal' (0.945 inches). The effect of this is that, with the eye at rest, the rays of light from distant objects do not bend in quite enough to make a clear picture on the retina. If, however, the person accommodates (i.e. increases the curve of the lens by means of the ciliary muscle) the rays of light will be bent more, and a clear picture will be formed on the retina. If now the person wants to read he has to accommodate a bit more still (and also, of course, converge or turn his eyes inwards to align them on the book), when near things become clear. As we saw before, the ciliary muscle is a very slick, strong, automatic and tireless little thing, so that the person who is slightly long-sighted and is young and healthy is quite comfortable and sees well at all distances. If, however, the eyeball is *very* short from back to front (as it is in some children before they have fully grown) the ciliary muscle may grow tired and the person will have a headache or an eyeache, or possibly will not be able to curve his lens enough to get the picture clearly focused on his retina. This is especially likely to happen in childhood

if the child has not yet gained proper control over the muscles which converge the two eyes and make them work together, and the effort of trying to accommodate and converge to the right extent may be too much and various troubles, such as squint, result.

On the other hand, longsight tends to become troublesome the older we get. This is because the lens loses its elasticity little by little every year until finally it will not curve enough during accommodation to make a clear picture. This is first noticed in reading and later will apply to distant objects as well. The age at which it is noticed depends on how longsighted the person was to start with (how much too short the diameter of the eye), since, roughly speaking, the loss of elasticity of the lens occurs at a constant rate in everyone. So far we do not know how to prevent this, any more than we know how to prevent old age. We do know, however, that continued close work does not hasten it and may even slightly retard it. People often think that they have 'worn out' their eyes when it begins, but this is quite untrue. The eyes, like every other organ, should be used if they are to remain healthy. This loss of elasticity progresses at roughly an even rate for the individual and at a fairly similar rate for nearly everyone in health. There is some evidence that the wearing of glasses makes us more critical so that we notice it more, and may think it has been caused by the glasses. Anyone of middle age who has just taken to glasses for reading knows that he can still read without his glasses if he does not mind holding the book rather far away and seeing the print slightly blurred. When he puts on his glasses he at once sees how it ought to look and so by the contrast becomes more aware

of his defect. This bit of psychology applies to all wearers of spectacles (for all distances) who see more clearly with their glasses on. Until they had their spectacles they did not know how clear it was possible for things to look and so they were not conscious of their defect. Once they know, then they won't put up with imperfection any longer, but most of them translate this into saying that things now look worse without their glasses than they did before. A little thought will show the fallacy of this.

This hardening of the lens with age is called presbyopia (old age sight). We shall be able to stop it only when we know how to stop grey hair, loose teeth, stiff joints, wrinkled skin and all the rest of it, and prolong our lives indefinitely. As science increases the expectation of life, fewer people die of injuries and diseases and more of old age, so that this inability to read becomes much more apparent. In the Middle Ages, when only a few people could read and write and the majority died before the age of fifty-five, nobody bothered much about presbyopia, but now when the average man of affairs becomes really busy at sixty, it is essential for people to be able to read at any age.

The age at which most people notice difficulty in reading is between forty-five and fifty. People with very short eyes and therefore long sight or hypermetropia notice it much sooner; the rare normal usually goes on to fifty or so, and the person with an eye longer than normal and therefore short-sighted or myopic can read without glasses for proportionately more years still.

This brings us now to the next most common type, those people whose eyeballs are longer than normal

from back to front and who are said to be short-sighted. What is their position? First of all, their defect is usually not there when they are born, since it is due to an overgrowth of the eye and so may be expected to appear slowly during adolescence. The effect of having an eye which is longer than 'normal' is that rays of light from distant objects are bent more than is necessary for that particular eye, and form the picture somewhere in the vitreous jelly in front of the retina, so that by the time the rays have reached the retina they have separated again and the picture on the retina is blurred. Therefore the person who is short-sighted can see clearly only objects which are near to him, since the rays of light from them are divergent and so the extra length of his eyeball does not matter. He can, of course, see objects farther away, but they have faintly fluffy outlines and lack detail. A short-sighted person can often recognize a friend across the road, but it is by his clothes and gait rather than by his features. Short-sighted people do not have to accommodate, since if they accommodate for distant objects these will become more blurred still, and near objects are clear to them, anyway, so that all the short-sighted person does is to keep his ciliary muscle relaxed and quiet always, and to converge his eyes when he wants to read. It is for some people difficult to converge without accommodating; this sometimes gives rise to a feeling of strain and discomfort, but many short-sighted (myopic) people can read for hours without glasses and without discomfort, since in doing so they use less muscular effort than the 'normal'. In addition; it is obvious that since the curve of the lens need not be much altered, the hardening of the lens does

not matter and the myope can read without glasses up to any age, though he will have to hold the book closer to his eyes the more myopic he is. Such people, if they live to ninety or a hundred, usually get an interview in the daily press in which they state with pride that they can read the smallest print without glasses. This sounds miraculous to the long-sighted reporter, who never thinks to ask if they wear glasses for the cinema or if they can read bus numbers at 200 yards. Hence also the popular idea that a short-sighted eye is a 'strong' eye.

Short-sighted people therefore seem to be cut out by nature for a life of close work, and they practically all choose it for themselves. They are the ivory carvers, the illuminators of manuscripts, the miniature painters and students of the past, just as the long-sighted ones are the hunters, sailors, and men of action. Now that we can all take up any kind of work and can see at all distances with the appropriate glasses this distinction is no longer so obvious, but even now the short-sighted person tends to be a book-worm and the long-sighted one keen on games and sport.

Since the short-sighted eye is usually larger than normal it follows that it is very unlikely that a new-born baby will be short-sighted, and very likely that the short sight will begin to show itself during school life and will increase slightly all the time the child is growing. Since we know no means of checking the growth of the child (except by depriving it of certain vitamins) we know no cure for myopia, but when growth stops the lengthening of the eye stops too, and the sight need not change further. In a few rather rare instances the eye does continue to enlarge after

the growth of the body has stopped, and then the short sight goes on increasing throughout life and may become a serious handicap. Such eyes are less tough than normal and more vulnerable. So there are really two kinds of short sight, one, very common, in which a perfectly healthy eye has grown a little too big, and the other rather in the nature of a progressive disease. The latter is rare, the former fairly common and usually inherited. Certain families tend to be short-sighted, and others long-sighted, since shape and size of the eye are inherited just as are shape and size of nose, colour of hair, etc.

There is another error of refraction, the name of which everyone knows, but the nature of which very few people really comprehend—astigmatism. This word, which has a more sinister sound than it deserves, is derived from two Greek words, 'a', meaning 'not' and 'stigma' meaning a 'point' or minute spot. It is applied to this error of refraction because here the rays of light entering the eye are *not* all brought to a focus at the same *point,* some of them joining each other further back than the others. The cause is commonly an irregular curve of the corneal surface, this being flatter in one direction than another, instead of being a perfectly spherical curve. The eye as a whole may be long or short-sighted and in addition is flatter in one meridian than another, so that even were the long or the short sight corrected by glasses the picture still would not be sharp. It is possible by using a cylindrical (as distinct from a spherical) lens in spectacles to correct this and bring all the rays of light neatly together again. In nearly everybody the cornea is a little more steeply curved from top to bottom than it is from side to side, and it

is very rare to find anyone with a perfectly spherical cornea. Therefore a small degree of astigmatism is present in practically everyone. In some animals (for example, seals) it is very marked and is actually of use to them in helping them to see both under water and in the air. In us it does not seem to serve any useful purpose and is sometimes an annoyance. It is, however, not a disease.

These errors of refraction, namely, long sight, short sight and astigmatism, are thus all really due to varieties of shape of the eyeball. The eye in all its essential features (i.e. clear cornea, lens and vitreous, normal retina, optic nerve and brain) is perfectly healthy. All that is wrong is that the rays of light do not easily come to a sharp focus on the retina. If nothing is done about them all that happens is loss of efficiency and often extreme discomfort, but never blindness, since the fact that the picture is blurred cannot harm the retina any more than a wrongly focused camera can do anything to a photographic film other than produce a foggy picture. Even if the trouble is over-action and fatigue of the ciliary muscle, this will not blow up or vanish away. It will only get very tired and need a rest to recover.

Although errors of refraction lead to much bother in modern life, an equal source of trouble is disorder of function of the muscles which move the eyes in the head, so that something occurs which upsets binocular vision. We can easily see how essential it is for the eyes to be both directed straight towards the thing we wish to see. Try this. Look at any object, say the clock on the mantelpiece, with both your eyes open; press your fingers gently on your right eyeball through the upper lid towards its outer end, displacing the eye-

ball a very little downwards. Immediately the clock will appear double; you will see two clocks, one above the other and usually overlapping a bit. This is because, owing to the displacement of your eye, the rays of light from the clock no longer fall on corresponding points (your two maculas) in your retinas. You have only displaced your eye a very little, yet the separation of the images of the clock is very evident. Try another experiment. Continue to look at the clock, but this time, instead of pressing your eye, hold your finger up in front of you about a foot from your eyes and in line with the clock. Keep looking hard at the clock. If you have full binocular sight, the finger will appear double (one on each side of the clock). This is because, since the picture of the clock is occupying both maculae and the finger is not in the direct line of sight from either eye to the clock, the picture of the finger will not fall on corresponding retinal areas in each eye, so we see it double. Some people find this trick difficult to do, and some are annoyed at being asked to do it because 'seeing double' is associated in their minds with alcohol. They remember the old cure for a cold—'Go to bed, hang your hat on the end of the bed and drink hot toddy till you see two hats.' This is dangerous advice, since the person with no binocular vision and the person with perfect binocular vision will never see two hats however long they go on drinking. Many people will, however, since in them the muscular balance between their two eyes is present, but not very strong, so that their eyes easily get out of alignment when they are tired or when alcohol has lessened their muscular control. This condition is called a latent squint, which brings us to the question: 'What is a squint?'

Some people use the term 'squinting', as we explained above, when they simply mean screwing up their eyes. This is medically incorrect. A person squints when one eye is directed towards the object looked at, the other being trained elsewhere, either too far towards the nose (convergent squint), or too far to the side (divergent squint), or up or down. There are all sorts of causes of squinting. If anything goes wrong with one of the muscles which move the eye so that it will not pull properly, then the two eyes will not be in proper alignment in all positions, a squint will result and the unfortunate patient will see double in the same way as you saw two clocks on the mantelpiece when you pushed one eye out of alignment.

This, however, is rather a rare happening. The commonest form of squint is that seen in young children, and this is peculiar in that the child hardly ever complains of seeing double, and, indeed, rarely complains at all. This seems strange, since an adult whose eyes had suddenly got into the squinting position would be driven distracted by the doubleness of everything. Children, however, are very adaptable, and quite soon the squinting child finds out how to ignore what it sees with the squinting eye. Those of you who have learned to look through a microscope keeping both eyes open know that it is quite possible to concentrate one's attention on the microscope slide seen with one eye, so that the extraneous objects seen with the other do not obtrude themselves at all. This is virtually what a squinting child does all the time. Some children use first one eye and then the other alternately, temporarily ignoring the other, but most children who squint keep one eye only for use and

continually ignore the other. This leads to a curious condition in which finally the squinting eye (the one which is ignored) cannot be used even if the child wants to; the ignoring of the picture has become permanent. This is a serious condition, but fortunately can be cured, though the cure is often very trying for the patient and its parents. The eye which is habitually used must be kept covered all the time so that the picture in the squinting eye becomes gradually as it were forced on the child's attention. This often takes many months, the child protesting for most of the time, since it does not want to make the effort necessary to recognize the picture in the eye it has got into the habit of ignoring. The treatment is well worth persevering in, however, provided the child is not too old. The older it is the harder to undo this ignoring, or, as it is usually called, 'suppression'. In adults it can never be accomplished unless they have the misfortune to lose the good eye, and very rarely even then, since a faculty never used always deteriorates in the end. The child who squints usually has an error of refraction as well, which is often indirectly the cause of the squint, since the child, unable to see clearly, may develop the squint in a misguided attempt to accommodate and converge harder in order to see more clearly. Most squints are curable provided treatment is started just as soon as the squint is noticed. It is most unwise to wait till the child is older in the hope that it will outgrow the squint. Indeed it is safe to say that from the age of six months any child with even a temporary deviation of an eye should be seen by an eye doctor. The earlier the treatment is begun the greater the chance of producing two normal eyes and binocular vision in the end. The treatment may

take several years, but is finally rewarding in most cases. Even if the squint disappears without treatment, the child may never develop binocular vision and will have a life long disorder of co-ordination between the two eyes. In many families the disorder is hereditary.

There are many causes of squint and of latent squint, many of them so far not understood. Actually, we know far more about the treatment of squints than their causes, and the causes of latent squints are even more obscure. In latent squint the eyes can be held in the proper position by an effort, so that the person does not see double, but suffers from headaches in the struggle to avoid an actual squint appearing.

One of the commonest forms of latent squint is what is known as convergency deficiency. The eyes are straight and comfortable when looking at a distant object, but when a near object is looked at (e.g. in reading) the effort required to keep both eyes converged on the page is greater than normal and a sense of strain is felt which often makes reading so uncomfortable that the patient gives it up altogether, even though the print is quite clear and there is no difficulty in seeing the actual words. Many people with this condition get very frightened and may fear they are going blind. There is no need to worry, however, because of all the disorders of the eye movements it is the most easily cured and its cure brings great relief, since the patient is able to read again in comfort, often after many years of strained feelings. The treatment consists mostly in developing a greater amplitude of convergence than is normally required, i.e. teaching the patient to over-converge as it were, so that he builds up a large reserve. Some people fear

that treatment will make them squint because they have no idea of the distinction between squinting and convergence. In squinting, as we have seen, only one eye is aligned on the object looked at; in convergence both eyes are turned inwards, in order to be both aligned on a near object. A person who is looking at a word in small print, but whose eyes are straight and parallel, is really squinting (by definition) because the image of the print does not fall on corresponding points in his two eyes. In order to make it do so he must converge them both.

We now come to the question of spectacles and why we wear them. The ancients did not wear spectacles, though they knew of magnifying glasses. These they did not use for reading, because in that spacious age the old men kept young slaves to read to them, unless they had the luck to be short-sighted. Who, then, invented spectacles? Who is responsible for the vast modern organization of optical glass manufacturers, gold and tortoiseshell workers, plastic chemists, lens grinders, opticians, ophthalmic surgeons, factories, shops, clinics, eye hospitals and consulting rooms, which has arisen from them? We are so used to these things and find them so much a part of modern life that we do not often stop to remember that neither Plato, nor Aristotle, Buddha nor Mohammed, Charlemagne nor William the Conqueror ever wore spectacles, and yet the world went on in much the same way, philosophies, religions and wars included, as it does now. One big difference to-day is, of course, the much higher proportion of the population that can read and write and therefore demands good sight. Another is possibly the longer expectation of life. It is, however, obvious that spectacles are not an essen-

tial part of civilization, though undoubtedly a desirable one, as anyone who has lost his reading glasses will readily admit. Who, then, began it all? We do not certainly know. In a church in Florence is an almost unknown tomb with the inscription 'Here lies Salvino degli Armatis, the inventor of spectacles. Died 1317. May God pardon his sins.' Whether we echo that sentiment or not will depend on the state of our eyes and on our attitude towards glasses. We need not, however, restrict our curses or our blessings to Salvino the Italian, since equally responsible with him stand an Englishman, the famous friar, Roger Bacon, who, together with John de Peckham, Archbishop of Canterbury, was responsible for many of the first experiments in optics and for the first serious study in England of the laws governing the paths of rays of light, and of perspective.

At first, spectacles were balanced precariously on the nose, or held in the hand as a lorgnon, or tied to the ears with thread or string. From the fourteenth to the eighteenth centuries the frames (similar to ours) were made mostly of horn. In the nineteenth century gold frames and rimless glasses, unequalled for lightness and comfort, appeared, but the decadent twentieth century has reverted to the mediaeval horn and tortoiseshell rim again, exactly why is not certain, but possibly owing merely to the skilful cultivation of a fashion, to the high price of gold, and to the rise of new industries, the trend being at the moment strongly in favour of synthetic plastics.

People have to wear glasses for three reasons; first, to make them see better (either in the distance or for reading, or for both); second, to relieve headaches and the sensation called 'eyestrain'; and third, as part of

the treatment of squint. People should not wear glasses to correct an error of refraction just because it is present, and for no other reason. If they ought to, then 98 per cent of the population would be in glasses, which is manifestly absurd. There are, however, a large number of people who hold that to be less than perfect is a pity, and so maintain that any error of refraction must be corrected merely because it exists and irrespective of whether it causes symptoms or reduces the acuity of sight. The decision as to whether a given error of refraction in a given patient should or should not be corrected with glasses is often quite a difficult problem and demands wide medical experience. After all, an error of refraction can cause a headache, but there are twenty-three other possible causes, so that to correct the error of refraction which you can find in 98 per cent of all patients with headaches is a very lazy way of finding out whether the two are connected. If the headache is due to one of the other causes it won't disappear when the patient wears the correcting glasses, and you then have to start all over again to track it to its source. An optician is skilled in correcting errors of refraction, but, not having had the six years' preliminary training as a doctor that an ophthalmic surgeon has had, is not to be expected to be able to differentiate eye headaches from others, and so will order glasses much more often than they are really needed. We had reason recently to examine a group of twenty-eight girls working in a factory on fine work. They all possessed excellent eyes and got no headaches from the work. Eleven of them had spectacles which they kept in a drawer on the bench, but never wore. They had bought them thinking it advisable to correct their

small errors of refraction and had then discovered that it was not necessary, as they saw excellently and had no discomfort without. This sort of thing often happens. The patient thinks he is cured of the error of refraction by wearing the glasses for a time, but actually what has happened is that he has discovered how to adjust himself to his error and live with it in peace. People often think that by correcting small, symptomless errors they in some way 'preserve' their eyes. There is no foundation for this, since wearing glasses cannot alter the shape of the eye or the elasticity of the lens, which really matter. They are merely optical instruments to focus rays of light on the retina when we cannot do it by ourselves with ease and comfort.

Let us now consider various kinds of eyes in the heads of various kinds of people and decide which of them should wear glasses.

Take the children first. In childhood, as we have seen, there is often excessive long sight, because the eye is not fully grown and there may be a squint because the child has not learnt to control the position of its eyes. Glasses are required as part of the treatment of squint, since by making it easier to see, they lessen the tendency of the eyes to get out of alignment. All children who squint should have their errors of refraction corrected, but if the squint is cured (perhaps by a combination of glasses, exercises and operation) and the eye grows to the normal size, the glasses may be dispensed with later in many cases. Children just learning to read may develop headaches because they have to accommodate more and for longer periods than they have had to do before. Glasses help this effort while the eye is growing and

can later be given up when development is complete. This temporary use of glasses is often necessary only because we teach children to read before their eyes are fully grown and before their binocular control is established.

There is another curious condition found in children for which the parent or teacher thinks glasses should be ordered, and therefore takes the child to an ophthalmologist. He usually surprises them by saying there is nothing wrong with the child's eyes. 'But,' says the parent, 'he can't learn to read because he sees words backwards and letters sometimes upside down!' The condition is called congenital word blindness or congenital alexia and is not easily explained. In it the mental appreciation of right and left and up and down and so on is very slow in developing, so that the child, seeing no difference between in shape between p and d and q and b but only a difference in position, finds it very hard to remember which is which and so apparently often reverses them. At the same time the power to synthesize the seen letters of a word into the sound of the word is imperfectly developed, so that, though the print is seen quite clearly, it will have very little meaning. Such children are in no sense mentally defective or slow learners but have a genuine difficulty which can only be overcome by slow patient individual teaching by a teacher who understands their trouble. They are often very good at arithmetic because each word is represented by a single symbol. It is obviously easier to learn and understand '8' than 'eight'. The condition rights itself in time, usually by the age of 12, unless the unfortunate child has been misunderstood and scolded. Then he may get so nerv-

ous about reading and acquire such a hatred of it that it is very difficult for him to learn, even later.

In the young adult with a moderate amount of long sight, and good binocular vision, glasses are hardly ever necessary. At a varying age, however, depending on the amount of long sight and the kind of work, glasses become necessary for reading and sewing, etc., because the person cannot accommodate hard enough without discomfort. Later, as long-sighted people get older and cannot accommodate at all, they must wear glasses all the time if they wish to see clearly, one kind of distance and another for reading, since they cannot now alter their focus themselves. Provided the retina is healthy and the lens clear the person will see properly with glasses up to any age, as all that the glasses do is bend the rays of light in such a way as to make a clear picture and discount the erroneous shape of the eye.

Now take the short-sighted person. If a child is born short-sighted it will make no complaint. This is often not realized, but is obvious if we stop to think. The child has no criterion by which to judge the normal. If it has never seen distant objects clearly it will never realize that other people can. The exclamation of a small short-sighted child given glasses for the first time and sent for a walk shows the state of mind. She said, 'Look! Do you know that a tree is made of leaves?' She had always seen the tree, but as a sort of mass of green cotton-wool. The revelation of its detail enchanted her, but she did not really think her long-sighted companion without glasses could see it, too.

A child who develops short sight fairly early in school life and is allowed to grow up without glasses

acquires usually a distinctive and slightly peculiar character, since it is cut off from and simply does not understand many of the things that help in the development of a normal child in a long-sighted world. First of all, it is intensely interested in books and in all fine detail and very bored with games. This is understandable, as if you cannot see a ball till it is within three yards of you, you have very little chance of catching or hitting it accurately, and if you are no good at games you cannot be expected to be interested in them. You can, of course, read for hours without effort and so tend to live in a world of books and fantasy, in which you wander dreamily about, getting a name for standoffishness, since you pass your friends in the street and never bother to pay much attention to the real world. This looks rather smooth and picturesque, but is stripped of significant detail. Such children win scholarships and may be correspondingly unpopular. If they are not given glasses they drift into jobs entailing close work, they get round-shouldered and peer at their work. They get wrinkles at the corners of their eyes because they are always screwing up their lids to try to make a sort of 'pinhole' camera to help them to see distant objects better, and this gives them a feeling of strain. They often have latent squints also. When finally they decide for themselves that they ought to have glasses they are often disappointed and say they cannot wear them. This is because of two things. The first is well explained by the following conversation which is fairly common:

Patient: Doctor, I think these glasses are wrong. I can't wear them.

Doctor: You seem to be able to see quite clearly with them. What is wrong?

Patient: I can't bear it. They make all my friends look so ugly. They all have spots on their faces and their collars are dirty, and all the houses look so untidy and old. I don't like it. I don't *want* to see all that detail.

All one can say is that it is a pity such a patient was allowed to grow up so out of touch with reality, as this attitude of not wanting to see what is ugly may extend to one's attitude towards life and lead to a deliberate shelving of responsibility.

The second thing which leads to disappointment when glasses are first worn is the difficulty at first of judging distances accurately. This is because the patient thinks the object is nearer than it is, because he now sees it more clearly than he used to do. This soon disappears if the patient will wear his glasses all day to start with.

It is therefore necessary for the myopic child to wear glasses practically all the time. If he does not, he may develop the qualities described above, which in a world arranged for long-sighted people is not desirable. Secondly he will get into trouble at school, as he will copy inaccurately from the blackboard and will be scolded for appearing stupid over things he has not been able to see. Also, the screwing up of the eyes in trying to see and the latent squint that often accompanies myopia will lead to much sense of strain, discomfort and often headache. The derivation of the word 'myopia' has reference to this habit of screwing up the eyes, which was recognized by the Greeks as undesirable, and the figurative use in such phrases as 'a short-sighted policy' makes clear that in the judgment of the majority myopia is a mistake. We should not therefore subject our children to this condition

uncorrected. Indeed, there are other medical reasons connected with the factors influencing the growth of the eye and the proper use of the ciliary muscle and the external muscles, which make it essential for glasses to be worn during the growing period. When the myopia has become stationary (growth of the eye has stopped) the patient can please himself. He can take off his glasses when he does not want to see and wear them when he does. In other words, glasses should be considered as an optical instrument for use when we want to see, like an opera-glass or a microscope. There is nothing magical or curative about glasses. All they ever do is to bring the rays of light to a focus on the retina. The more accurately they do that the more comfortable they are to wear and the better worth wearing, but even if they are quite wrong they cannot injure the retina. Indeed, a famous 'unorthodox' treatment in Germany before the war was to force the patient to wear quite wrong glasses through which he could hardly see, so that when he took them off he got the impression that he was now seeing much better than before. This was rather derogatory to the patient's intelligence, but never actually harmed the eyes, any more than looking through frosted or wavy window-glass would do.

Many people are afraid of what 'strong' glasses will do to them. Whatever the lens required to bring the rays of light to focus on the retina may be, it is neither 'strong' nor 'weak' for that individual. It is correct. To wear a less correct lens would obviously be silly. A less correct lens might well be described as 'too strong', or 'too weak', and so would be uncomfortable, but the correct lens for that person is neither 'strong' nor 'weak'. It is that which produces the sharpest

possible picture on the retina with the accommodation relaxed and so enables the patient to see the most clearly with the least effort. Unless you are aiming at this, it is silly to wear glasses at all.

Some people think highly curved lenses are ugly, and it is true that people with very high errors of refraction can now wear contact lenses instead of spectacles if they have the luck to find a skilled fitter, which may be difficult. There has been much talk of these invisible glass shells, which fit on the eye under the lids, but there is a lot of loose thinking about them. They are not substitutes for spectacles, but are primarily used to correct errors of refraction due to a wavy or irregular corneal surface, which cannot be corrected by spectacles. For this they are extraordinarily good. They are occasionally used instead of spectacles if the patient is exceedingly short- or long-sighted, but for all the common low-grade errors they are really of very little use.

So we see that all myopes should wear glasses, that all long-sighted people and presbyopes should wear glasses if they cannot see without them, and that many people with squints and latent squints should wear them while undergoing treatment.

The question now arises, 'What about the numerous young adults who wear glasses for headache and eye strain and yet can see perfectly well without them? Could they be cured?' The answer is that a great number of them could be cured of their symptoms, though none of them could be cured of their error of refraction, since, as we have seen, this is structural, and does not matter if it does not produce symptoms. There are three types of spectacle wearers who quite possibly wear them unnecessarily. They

all have a small amount of long sight and often slight astigmatism, but they all see quite clearly without glasses and yet are uncomfortable if they do not wear them. In one group there is a defect of muscle balance or binocular vision, in another there is headache, more or less of the migraine type, and in the third there is a psychological disturbance, usually an anxiety or fear. Often these three things co-exist, and it is a long and difficult process, suitable only for a skilled doctor, to disentangle them. In a word, the muscular imbalance can be cured by exercises, the headache can often be relieved by investigating and treating the general health (remember the twenty-four possible causes of headache), and the neurosis can be removed by getting the patient to face his fears and conflicts consciously and see them for what they are.

This brings us on to the question of exercises. There are several kinds of eye exercises, and as we have seen that there are various reasons for not seeing clearly and comfortably, so we must realize that some eye exercises will be good for one thing and some for another. No system of eye exercises applied indiscriminately to all cases can have any sense in it, any more than one or two Swedish exercises can cure any muscular weakness whatever. We do not do arm and trunk exercises for flat feet, nor do we do tiptoe exercises for a stiff shoulder. Many 'systems' of eye exercises are as silly as this, and none is any use unless the patient has been thoroughly examined to see what is wrong, that is, whether the two eyes do not converge or whether one converges too much, or whether the ciliary muscle does not contract, and so on. Once this has been disentangled by the ophthalmic surgeon he can prescribe the appropriate exercises.

It must, however, be thoroughly realized that no exercises can alter the shape of the eyeball and change the refraction. What exercises can do is to improve very markedly the use we are able to make of the eyes we have got.

Practically all effective eye exercises aim at increasing the precision with which the patient can turn the two eyes together on to a given point and see it stereoscopically. Such exercises are called 'orthoptic' exercises, and they are very valuable in the treatment of all kinds of squint, of reading difficulties due to convergence deficiency and of conditions where the judgment of distance is poor. The diagnosis is first made by an ophthalmic surgeon and the exercises are carried out under his direction by a trained orthoptist. On no account should eye exercises be practised indiscriminately without first having the defect sorted out. People with only one eye need never do eye exercises, as they obviously cannot get any form of squint.

Apart from orthoptic exercises, which can be obtained on the recommendation of a doctor at various clinics throughout the country, there exist a number of unscientific systems. These are given indiscriminately to all comers and no preliminary examination is made to decide what is wrong. Most of such exercises are harmless and ineffective, such as staring at a coloured light, opening and shutting the eye so many times a minute, rolling the eyes from side to side, swaying the body rhythmically, closing the eyes and meditating on blackness, and various other suggestive performances which do not really exercise the eyes in precision of movement at all, but make the patient feel that something is being done. A large number of people

claim cures from these capers, but when the cases are investigated it is always found that the patient's eyes have not altered, but his attitude towards them has. He is no longer afraid to use them and no longer minds not seeing quite normally. What was really the matter with him was a functional disorder (see p. 160) in the course of which he had allowed his eyes to become his masters, and not his servants, and so was unable to make use of them when he wished. Any procedure, however illogical, if applied with faith and conviction, would have cured him equally well, but the cure would have been more lasting and fundamental if it had included a realization of what had really happened, and this it never does. The patient to-day, just as in the Middle Ages, demands magic, and would rather do anything than use his critical faculty and his common sense.

If the matter rested there not much harm would be done, but unfortunately, owing to ignorance and to the above-mentioned desire for the simplicity of the magical cure, a number of people who should have other treatment spend much time and money on exercises, and a certain number actually worsen their condition by these means. For instance, some systems used to include an instruction to train oneself to stare directly at the sun. If this is persisted in by a normal individual it will seriously and permanently impair his sight, though a patient with corneal scars can often do it with impunity, because the scars prevent so much light getting through to the retina. Again, a reading exercise is sometimes described in which each part of every letter in a word is to be minutely examined when reading, although it is well known that the most fluent readers look only at the general shape

of the word and do not consider its constituent letters at all.

There is a danger that patients with serious yet treatable disease such as glaucoma may waste valuable time on such procedures, and that children to whom a pair of glasses for short sight would make all the difference at school may be made to spend hours doing exercises which from their nature can have no effect on the shape of the eye.

Obviously if the trouble is a disorder of the way the eyes are used exercises will be valuable, but if it is a structural defect they will not help. The moral is obvious; first find out as exactly as possible what is wrong, and then decide on the treatment. To treat without diagnosing is as silly as pumping up a flat tyre without finding out where the puncture is.

CHAPTER XIII

Facts and Fancies about Ocular Hygiene and Spectacles

In a country in which everyone knows how to read and everyone is free to rush into print, it is not surprising that great confusion exists in the minds of most people as to which of the many conflicting statements presented to them is most worthy of belief. Is this the moment to Eat Less Bread, and Eat More Potatoes, or is it the other way round? Should one, having cleaned one's teeth with Blanco Champer's Hygienic Tooth Paste, bathe one's eyes with Forty-winks Fortifying Eyewash, or not? Should one Save Fuel by changing all the electric lamps to lower power, or protect one's eyes from strain by reading only with Goodchild's artificial daylight lamp? Should one go out to stare at the sun, or buy a pair of Lido's dark spectacles? Should one read in bed? Should one live on carrots? It is all very muddling!

Let us consider 'weak eyes'. Many children get sore and ulcerated eyelids and styes if their health is poor, their surroundings not satisfactory, their diet insufficiently varied and the right germ present. This condition is often called loosely 'weak eyes'. This term popularly includes sore lids, watery eyes and bloodshot eyes, none of which really has anything to do with the eyeball itself. It is not very logical to expect a pair of spectacles to kill the germs which are infect-

ing the roots of the eyelashes if you do not pull out the infected hairs and put some antiseptic on the lids. The glasses may, or may not, be necessary, but the need for them has nothing to do with the presence of the inflammation of the eyelids. A very old remedy for 'weak eyes' was to pierce the ears and wear ear-rings, the occasional success of the treatment possibly being due to infection of the hole in the ear-lobe with the same germ as the eyelids. This would act like an inoculation, just as the germs from our throats and noses are used to inoculate us against colds. Very old readers may remember the use of a seton* in the neck with the same object.

The routine use of eye lotions is a point of great public uncertainty. Some people think that the eyes should be washed out daily with an eye-bath, just as one cleans one's teeth. This is a mistake based on ignorance of the way the eye normally protects itself from infection. In health the conjunctiva (the thin, moist skin over the visible part of the eyeball) is kept wet and free from germs by the thin film of tears which is always being secreted by the glands. This clear fluid film is antiseptic and acts both mechanically by washing away germs and dirt and chemically by rendering them harmless. This natural protective mechanism should never be interfered with unless it has gone wrong. If you are continually washing it away you will dilute the tears, irritate the conjunctiva and render your eye more susceptible to infection. Often patients come to eye hospitals with a story that they thought they had conjunctivitis and so bought

* A 'seton' is a piece of thread, threaded through the skin and underlying tissues with the object of producing a running sore. It is a completely obsolete form of treatment.

some eye lotion which they have used (often changing the brand several times) for months, with little or no relief. The doctor then says, 'Do not touch your eyes for a week, as I want to try to find the germ which is causing this, and it is no good hunting for it if you are doing any treatment.' The patient often comes back in a week and says, 'Thank you, I have now recovered. I got better as soon as I stopped treating it.' Possibly when the patient started treating his eyes they really were infected and the lotion cleared this up; continued routine use of the lotion, however, again irritated his eyes so that his last state was little better than his first. Eye lotions and ointments are of undoubted value, but it is as necessary to know when to stop using them as when to start.

There are many types of eye drops and eye ointments and eye lotions, many of them very useful and some of them essential in certain cases. Let us see why these things are used. First of all, eye drops and ointments are used to disinfect the conjunctiva and lids, but these vary with the germ or bacterium it is desired to kill. There is no universal eye antiseptic. The doctor must decide which germ it is and then order the appropriate drug which kills that particular bacterium. You do not set mousetraps for cockroaches, nor hunt tigers with rat poison. You decide what you want to kill and use the weapon most likely to succeed. If there is no enemy there is no need to do anything. Occasionally a hot sore feeling in the eyes, with slight redness, is due to something wrong with the patient's habits or diet. Excessive exposure to cigarette smoke or other irritating smokes or dusts will produce it, and, rather more rarely, a dietary deficiency (especially too little vitamin B_2—present in fresh meat, eggs,

liver, yeast, yellow vegetables and wholemeal flour). It would obviously be silly to expect an eye lotion to cure this sort of thing.

Secondly, eye drops are used for their effect on the size of the pupil. Some drops make the pupil very large and stop the ciliary muscle acting; they are used in the treatment of inflammations and for purposes of examining the eyes. There are other drops which make the pupil very small and tone up the ciliary muscle. These are used in conditions where there is too much aqueous and vitreous and the pressure inside the eye is raised and the eye tense and hard. Never put into your own eye the eye-drop intended for another. This sounds self-evident, and yet a number of people every year come to eye hospitals complaining that they cannot see clearly and that this followed the use of the baby's ointment, or grandmother's eye-drops. These drugs are used for their effect on the mechanism inside the eye.

Now as to using the eyes and the kind of light in which to use them. First, given a healthy adult eye, it is impossible to wear it out by over use. The eye is in use and its muscles are working every second of our waking lives, but it shows normally no signs of wear and tear other than the hardening of the lens, which happens anyway, and possibly rather earlier if the eyes are *not* used. Similarly we cannot wear out our ears with listening. We cannot even shut our ears, so that they have to function in the dark, too, yet you never hear of 'ear strain', or that such and such a musician has worn out his ears! Many people, as they get older, get slightly deaf, and yet no one imagines that they could have prevented this by 'resting' their ears throughout their lives. Neither does our sense of

touch deteriorate through use. Indeed, it rather improves, and so on with our other senses. There is no reason to fear that our eyes will behave differently or that they are inherently more delicate than our ears. There is, on the other hand, some evidence that eyesight improves with use. If this were not so, there would be no point in doing exercises to improve one's binocular vision, or to restore sight to a disused eye in squint.

People suffer from 'eyestrain' (which means discomfort, pain in the eyes, or headache, but not inability to see) on doing close work, rather than when they lead outdoor lives. It occurs only if one is able to see to read clearly. There is no eyestrain if one cannot anyhow focus properly, since there is no point in making any effort. So when people complain of eyestrain usually one of three things is the matter; either they are so long-sighted that they cannot focus properly without the ciliary muscle getting tired, or there is a disorder of binocular vision so that they find it difficult to keep both eyes on the print together, or there is a psychological reason why they are *afraid* to use their eyes. There is no treatment for the first condition except glasses, since we cannot alter the shape of the eye or increase the elasticity of the lens, but usually the glasses need be worn only when one wants to read, not necessarily all the time. The second condition can be treated and very frequently cured completely by training the patient to gain a better control over the movements of his eyes. The third condition can be cured by understanding treatment, both by someone with deep insight into the patient's difficulties, and also by 'magic', i.e. very strong suggestion through some striking procedure which persuades the patient

that something extraordinary is being done at last, and so restores his lost confidence.

In all cases it should be realized that a pair of spectacles is just as much an optical instrument as a telescope or a microscope and should be looked on in this way, and not, as many do, as a peg on which to hang all one's discontent and lack of confidence. Since it is possible now to make spectacle lenses extremely accurately and frames of a perfect fit, far more people tend to wear them now than in the past, when being heavy, uncomfortable, and precariously balanced, only those people who really could not see without them found it worth while to struggle with them.

You should know a few things about ordinary spectacles. There are four ordinary kinds of spectacles, namely, spectacles for constant wear, spectacles for distance only, spectacles for close work only, and bifocal spectacles.

Spectacles for constant wear: these are for people under the presbyopic age who are too short-sighted, too long-sighted, or too astigmatic to get on without them, or for children, as part of the treatment of squint.

Spectacles for distance only are for myopes (short-sighted people) who happen to be able to read in comfort without them.

Spectacles for close work only are for hypermetropes (long-sighted people) and presbyopes.

Bifocal spectacles are for presbyopes who wish to see both near and distant objects with the same pair of glasses. Some people find them awkward to wear, but if they are used intelligently they are a great boon.

Remember always when buying spectacles that a well-fitting frame is as important as a correct lens, because the lens will be correct only if it is set at the correct angle and at the right distance from the eye.

It is also important that you understand how to look after your spectacles. Never lay them down so that the front surface of the glass rests on the table or you will find that they become scratched. Always put them with the side pieces downwards. Also, always keep them clean and do not strain the little hinges by pulling them off with one hand so that they get out of alignment. Remember to have them tightened and adjusted at regular intervals. Children who wear spectacles should be taught to feel responsible for keeping them in order. If a child has to wear glasses for games it is a comfort to have the lenses themselves made of plastic so that they will be unbreakable. This has the slight disadvantage that these lenses scratch more easily than glass ones.

How about people who have only one eye? If they have always been blind or nearly blind in one eye they may never find it out. This seems almost incredible, but it frequently happens that a patient comes into an eye hospital saying, 'I have just gone blind in my left eye.' The doctor looks at it and is convinced that the condition has been there since birth and so he asks exactly what happened. The patient says, 'I got a bit of grit in my right eye and when I shut it and rubbed it I found I could not see with my left.' So unless you find it out by accident, you may never know. A person who has always had only one eye notices no disability. He can judge distances fairly well (using other criteria than stereoscopic vision)

and does not get eye strain (unless he is fairly long-sighted) because he does not have to try to keep both eyes on the book at once. Indeed, normal people, when very tired, sometimes find it easier to read with one eye shut. If, however, a normal adult loses an eye by accident he is naturally very upset and often thinks, 'Now my remaining eye will have to do double work.' This is not so. One eye does no more work than two, since all the eye can do anyway is focus a clear picture on the retina and telegraph it to the brain. What will, in fact, upset him is the loss of perspective and stereoscopic vision. Everything will look flat and he will have to learn other tricks to judge distances, tricks which the man one-eyed from birth has always known, namely, judgment by the relative size of objects and by their parallactic movement (see p. 14. That this can be done is undoubted; witness the pilots who have learned to fly successfully after losing an eye. The man who has lost an eye need never be afraid to use the remaining one. It will not wear out any quicker because it is alone, any more than an ear will deteriorate because its fellow is deaf.

All that we have said about eye strain applies to normal adults. The question of treating eye strain in children is a rather different job, as it is bound up with the question of the rate of growth of the eye and the age at which the child attains binocular vision. How then can we help and protect our children's eyes? First of all, a baby when it is born does not see clearly. Do not worry about its eyes for six months, unless it is obviously blind. Do not keep on showing things to the baby. Do not hang dangly toys just above its head or try to make it look. Leave it alone and feed it. It is a little vegetable at this stage. If, after six months,

it seems to squint, then have a thorough examination by an eye specialist at once.

Soon after six months the baby should begin to learn to turn both its eyes together on to what it wants to see. It will do this first when looking at distant objects and only much later when looking at objects close to it. We do not know the exact age at which full convergence and perfect binocular vision are established, so that both eyes focus together on an object whether it is near or far away, but probably it is not till the age of six years or so. Children of four are usually capable of being taught to read, but it is obvious that if they have not got the full power of convergence by then they should be taught with big letters or blocks with which they can play, rather than with small print in books which they always tend to hold too close to their eyes. Use any games you think of to stimulate an accurate judgment of distance; ball games, and indeed all throwing and catching games are good. If you have a stereoscope or one of those books of anaglyphs (which are red and green photographs superimposed which appear in relief when viewed through red and green spectacles) which were popular as toys a few years ago, encourage the children to play with them and try to find out whether they see the pictures standing out properly and looking solid.

When the child can read it should have a proper sight test, and again it should be tested some time between the ages of ten and fourteen, to make sure that short sight is not developing. Sight testing in children should always be done by a doctor and not by an optician, since the latter may possibly feel bound to order spectacles because he has found an error of refraction. Whether a child needs spectacles

or not depends on a variety of other factors, as we have seen.

Should children read in bed? This depends on the position in which they lie and on how long they go on. If there is a good light and the child sits up and holds the book evenly in front of the two eyes it is just the same as reading anywhere else, but to lie on the side and put the book on the pillow is not a good idea, as it is difficult to maintain binocular vision under these conditions. The real trouble, however, is that if reading in bed is allowed it is almost certain to go on for much too long and so deprive the child of necessary sleep.

Now we come to the question of lighting, of glare, and of the use of sun-glasses. First of all, people vary very much in their reaction to bright lights. Some people rejoice in them, others fear them. This is largely psychological, since only exceedingly bright lights can do any real harm to the eyes. We all know that light reflected off snow can cause a very painful condition (snow blindness) which takes a few days to pass off. This is due to the ultra-violet rays destroying some of the cells in the cornea and not the effect of the visible light on the retina. Indeed, winter sports addicts often find that they may get sore eyes on quite a dull day, when the sun is obscured. Similar troubles occur when the ultra-violet light is from an artificial source. Film actors may get it if they look at the arc lights in the studio, welders will get it if they do not use the protective screen when watching the oxy-acetylene flame or the electric arc. Soldiers fighting in deserts with glare from the bright sand and sailors staring at the sea in sunshine may also suffer great ocular discomfort unless they wear some form of

tinted glass. All these suffer from the ill effects of the light to the outside of their eyes, but there is another curious effect which climbers on snow sometimes notice, and also, curiously enough, people who are just beginning to see again after a cataract operation. This shows itself as a tendency for everything to look pink. It is called erythropsia, or red vision. Anyone experiencing it should diminish the light (go indoors or put on tinted glasses) and the effect will wear off. It will not do any permanent damage to the retina.

Apart from snow blindness and erythropsia, which are examples of the ill effects of too much visible and ultra-violet light, there is no golden rule for the amount of light which is good for us. Driving into the sun or watching glittering reflections on moving machinery or water can be very trying and will give many people a headache. Just before the war a fashion-induced craze for exotic tinted sun spectacles swept Europe. It was definitely not the thing to appear on the beach at Deauville or Juan les Pins without one's sun-glasses, quaintly rimmed in white or pale blue. It is true that certain people, more often the blond blue-eyed type, though not invariably, are made uncomfortable by bright sun, but they are certainly not so numerous as the fashion made us imagine. In England there is never enough ultra-violet light in any situation to be harmful, and indeed one can say as a general rule that only on high snow mountains, in tropic deserts, on southern seas, and in the drier parts of Australia is there actual need for sun glasses. In certain diseased conditions of the eyes they may be essential, but it is otherwise not a very good idea to wear them, since a vicious circle may be set up whereby the less light one lets into one's eyes the less

one wants to see, and so one tends to buy each successive pair of glasses a shade darker than the last until finally one feels that it is necessary to wear them even on dull days. The same thing applies to other patented types of glass which are used to make ordinary spectacles. They aim at cutting out the ultra-violet light (and sometimes the infra-red as well) without appreciably altering the visible spectrum., They are, in most temperate climates, nearly always unnecessary but may be helpful in special circumstances.

Trying to read or work in a poor light will produce a headache. Most interior domestic lighting, especially at the present time, tends to be on the dim side, but again people differ very much about the amount of light they like. Age and kind of work make a lot of difference. A presbyope trying to unpick a machined hem on a black frock at night will complain of the poor light while her husband reading a novel will notice nothing, and their myopic son studying for an examination may be sitting quite happily in the darkest corner of the room. Again fashion has led us into strange ways. It has imposed upon us indirect interior lighting and sometimes given us no alternative. While a source of light which shines in our eyes is extremely unpleasant, a diffuse light of low intensity reflected from the ceiling and casting very little shadow is almost equally annoying to work by. For ordinary reading diffuse light is all right if sufficiently bright, but for work such as sewing where a directional light casting shadows is required to bring out the texture, it is of no use. For that a reading lamp with an opaque shade painted white inside and arranged quite close to the work but not shining on

the eyes is most satisfactory. The ideal interior lighting should simulate daylight as much as possible. It should avoid glare and annoying reflections from highly polished surfaces in the vicinity of any work in progress. The illumination should be adequate for the particular task and the work should be slightly more brightly illuminated than the rest of the room. On the other hand, excessive contrast can be very trying. If in reading, for example, the book alone is brightly illuminated and the room very dark, discomfort and fatigue may result.

It is especially important that the intensity of the light be adequate, since visual acuity increases with illumination within certain limits. The ideal form of interior lighting has yet to be devised.

To sum up the care of the eyes for adults. We can say, firstly, make sure you have no disease of eyes, or body, or mind. Secondly, make sure whether you need glasses or orthoptic exercises or both or neither, and, thirdly, after that, leave your eyes alone; do not worry about them and use them as much as you like. Find out for yourself what kind of light suits you best. Do not smoke excessive shag or too many cigars and eat all the good food you can get. If you wear glasses they should be checked every three or four years. To this we would add, in the case of a child, do not be in a hurry to teach it to read; find out as soon as possible whether the child has binocular stereoscopic sight, and get it treated by an oculist if it has not.

Another set of misconceptions exists about the right person to deal with certain things which go wrong with the eyes. Many people think that chemists are specially qualified to get bits of grit out of eyes, that

general practitioners are the best kind of doctors to treat red, discharging or inflamed eyes, and that oculists and opticians exist solely for the purpose of ordering unnecessary and harmful pairs of spectacles. It is hardly necessary to point out that none of these statements is true.

CHAPTER XIV

The Blind People in England and Australia: why they are blind, what they can do and what we do for them.

When is a person blind? It is obvious that a lot must happen before anyone becomes totally blind, in the sense that they have no perception of light at all. Indeed such cases are relatively rare. What is more common is for sight to be impaired and not to be improvable by glasses or operation. Then we have to decide whether such people are technically and legally blind or not, because certain benefits are available to them if they are. How is this done? No one has so far found a completely satisfactory standard, so nearly every country has fallen back on a practical definition which makes no mention of the actual degree of sight present. The definition runs "so blind as to be unable to perform work for which eyesight is essential". This gets to the crux of the matter, namely the admission that the person is unable to work at an ordinary job and so must be helped.

Let us consider the various ways in which sight is affected in persons who are "legally" blind. Total blindness means that the person cannot distinguish light from darkness and is in the position of a creature, such as some species of ant, which has no light-receiving mechanism at all. Some technically blind people can perceive the difference between light and darkness (as our earthworm did in Chapter I), others, like the

marine worms, can tell the direction from which light is coming, others again can see vague shadowy outlines and patches of colour, others can see all around but have no macula so that there is a blind area in the centre of the field of vision and everything they look straight at disappears from view. Others again see a small patch of scenery very clearly but the rest is blank, as if they were looking down a long narrow tube. Again, if the retina is rucked up and full of holes, a patchy and distorted picture will result. Plate VIII shows the same scene as seen by technically blind people of these seven kinds. The scene is a house standing about 100 yards off with the sun shining from the right-hand side. The person is looking at the top right-hand corner of the front door. It is quite obvious that the behaviour of these seven blind men will be different in every case.

It is also evident, if we stop to think, that the condition and indeed the whole mental life of a person born with any of these types of visual imperfection will be different from that of one who has seen normally and has become blind. Let us consider the child born without any power of perceiving light. The channels of entry of knowledge about the world are hearing, touch, smell, taste and the proprioceptive sense. Such children early learn to pay a great deal of attention to the messages they receive through these senses and the amount of independence they ultimately acquire, if allowed to develop without the restraining hands of too solicitous relatives, is always astonishing. They should be allowed to run freely about and to fall over things and bump themselves and burn their fingers within reason and should on no account be hindered from exercising their curiosity. In this way they will

develop an extremely discriminating sense of touch and of the direction of sound, and will show the curious reaction called "the sense of obstacle", which prevents them walking into things.

It is this sense of obstacle which has been called the sixth sense of the blind. It is, as far as we know, an extreme awareness, cultivated by practice, of minute differences of sound (the sound of one's own footsteps), of temperature and of air currents. For example, if you are approaching a wall your footsteps will echo in a different way, the wind will be deflected by the wall and you may cease to feel it, and the wall may be warmer than the air, and even have a smell. All these things are heard and felt by sighted people, but they pay no attention to the messages they receive through hearing and touch, because it is so much easier just to look at the wall. What the blind do is to become aware of a host of things which we also experience, but habitually ignore. Usually this is done subconsciously by the blind and they cannot explain clearly how they know the wall is there. They do, however, usually say that a high wind or a fall of snow will throw them completely out, and they will lose their sense of direction. This may also happen if they get a cold in the head and become slightly deaf.

Such people, born blind, have no power of imagining what sight is like. They have no acute sense of loss, but rather a feeling of bored exasperation with the sighted world for spending so much time talking about things seen and for being so easily distracted from serious thought and conversation by passing sights. They pick up easily the use of visual symbolism in conversation, but have really no conception of the meaning of many of the words they use correctly,

such as colour, picture, outline, light, dark, misty, etc. They are often taken for walks by misguided friends who describe minutely the beauty of the trees, flowers and sunsets to them, to which they listen politely, concealing their boredom. As one such blind boy put it, "I don't really mind because I am fond of the friend I go for walks with and he enjoys talking about those meaningless things." To a blind person who has once seen, the situation is, of course, quite otherwise.

A person born with one of the types of defective sight described above will be less handicapped than a totally blind person, as he will find out quite a lot about the world with his imperfect sight. He will be able to make better use of it than would a normal person suddenly reduced to his level. He will have learned over a long period of time to interpret the very imperfect images he perceives by relating them to their objects by means of his other senses.

The condition of a normal person suddenly blinded is again very different. The shock is extremely great, and it will be a very long time before he can readjust himself to perceive and pay full attention to the messages he receives from his other senses, many of which, previously, he was in the habit of ignoring. The behaviour of such a person varies very much with his age and character. The younger he is when the accident occurs the greater the power of adaptation. Also the more intelligent the individual is, in most cases, the fuller will be his power of compensating for his loss. For instance, the power to read embossed type (Braille) with his fingers depends more on the person's desire to read than on his original delicacy of touch. Younger persons learn it more easily, it is true, but

even old people can sometimes manage it if they are sufficiently keenly interested. On cannot repeat too often the fact that blindness makes very little difference to a person's basic character and intelligence, but that these factors definitely determine his behaviour under the handicap of blindness. In one respect only is a change sometimes apparent and that is in the direction of improvement of memory.

If we want to go more deeply into why people go blind we can get a lot of information from studying the various forms which have to be filled up by blind persons on application for invalid pensions, for assistance, rehabilitation, education, and so on. In England and Australia and many other parts of the world these forms are available for study by doctors hoping to reduce the amount of eye disease.

In most civilised countries the principal causes of blindness are similar. In both England and Australia blind children are rare and they are now practically all blind from something wrong with the structure of their eyes (dating from before birth) and not from disease. In both countries the majority of blind persons are over 60 and are blind from various degenerative conditions or diseases due to age. Cataract heads the list in both. In many cases these persons are not permanently blind, as operation can restore their sight if the opaque lens is the only hindrance to seeing. Often, however, they are too old or infirm to stand surgery and often there is something else wrong with the eye as well as the cataract. Another old-age condition is senile macular degeneration, which means that, due to age, the central spot of clear sight, the macula, becomes as it were blurred and there is a patch of fuzziness on everything the patient looks at (see Plate VIII again).

This condition is the second commonest cause of blindness in England but is not so prevalent in Australia, being eighth on the list of our causes of blindness. Glaucoma (see page 174) occurs equally in both countries but severe degrees of shortsight are rarer in Australia. One black spot in the Australian records is the frequency of industrial accidents leading to blindness. In England, where safety precautions are taken much more seriously, the percentage of blindness from this cause is only 0.2%, while in Australia, with its happy-go-lucky philosophy and its tough-guy attitude to safety devices (protective goggles, machine guards, etc.), the percentage is six and a half times as much. In the United States, with its Wise Owl Clubs the figure is even lower than in England.*

In Australia, however, 2% of blind persons have had their sight destroyed by a disease not found in England but which actually is the commonest cause of blindness in the world today. This disease is trachoma (or Egyptian ophthalmia as it used to be called). It is an infectious chronic inflammation of the under surface of the upper lid which in the course of years spreads to the cornea and may make this opaque and so lead to blindness. It is fortunately easily cured if detected early before sight is affected. Four hundred million people in the world today suffer from it and at least ten per-cent. of them will become blind. Large international organisations exist to study and combat this disease and much is being done in Australia to try to eliminate it. At the present time it is found mostly among aboriginals and in white migrants from countries around the Mediterranean where it is rife. Its favourite

* The Wise Owls are workers who, by using a safety device, have survived an accident which would otherwise have led to loss of an eye.

breeding ground is (and has been for thousands of years) Egypt. Large areas of Africa, India, China, Japan and Russia are heavily infected and its eradication is one of the major Public Health responsibilities in any country which has a lot of it. It was common among white persons in Australia in the past but is disappearing as the hygiene of the people improves. Some old people still believe it is caused by sun, sand and heat, but it is really an infection with a virus. In the old days all conjunctivitis (inflammation of the thin moist skin covering the eyeball) was lumped together, especially in Western Australia, under the expressive slang term "sandy blight". Doctors now distinguish very many different types of infection and the term is meaningless.

There is another common eye disease in Australia which is not found in England but which, fortunately, hardly ever leads to blindness though its victims often fear that it will. This disease is called pterygium. It—far more than trachoma—is caused by sun, glare and hot, dry winds, which is why it is never seen in England. In this disease a triangular piece of inflamed mucous membrane encroaches on the edge of the cornea and extends on to it. This and, not as popularly thought, a cataract, is truly "a skin which grows over the eye". Minor degrees of it are common. It may have to be operated on.

So far we have been thinking about people blind in both eyes. How many people are blind in one eye we do not know. There must be a lot in England since the Trades Directory for London lists four firms engaged in the manufacture and sale of artificial eyes. The largest of these supplies 11,000 glass eyes a year, but these are largely replacements, since glass eyes "wear

out", become rough and scratched and get broken and so have to be renewed. Artificial eyes made of plastic are probably much more efficient; but even these become dull-looking after a time. This large firm has roughly six to eight new patients a week in London. No accurate figures are available but it is safe to say that many people are wearing an artificial eye unsuspected by their friends and largely unnoticed by people they meet.

There is a hospital story of a girl who had the misfortune to lose an eye, and who was not to be consoled and gradually fell into complete melancholy, thinking herself the most unfortunate of mortals. She was persuaded to accept the post of receptionist to a glass-eye maker and recovered completely in a week when she realised that everyone to whom she opened the door had lost an eye also and had survived. These people wearing glass eyes represent only a fraction of all people blind in one eye, since in many of them the blind eye has not been removed, or if it has, they do not wear an artificial one. If we add to this the number of people who at some time or other have lost work through an eye injury or disease, the importance of education, prevention and the supplying of adequate medical attention is obvious.

There are certain things that are interesting about the causes of blindness in different parts of the world because they reflect the different ways of life and the different standards of other countries. The Western way of life undoubtedly leads to a great reduction in blindness and eye disease generally, since in most European and Commonwealth countries only approximately one person or less in one thousand is blind. In Egypt in 1907 there were thirteen persons in every

thousand and 95 percent. of the total population suffered from some kind of eye disease, especially trachoma. The amount of blindness was roughly halved between 1907 and 1936. This was largely due to the building of more and better eye hospitals, to the education of the people to awareness that treatment was obtainable, and to the stamping out of such diseases as smallpox, which is a common cause of blindness in countries where it still rages but almost unknown in England and Australia. Roumania, which is a sort of junction of East and West, shows a marvellous example in this respect. In 1920 there were 20,580 cases of smallpox, many fatal, and 615 of them blinding the patients permanently from scars on the cornea. In 1933 six cases only of smallpox occurred in the whole country, and no eyes were lost. This was, of course, due to the introduction of vaccination.

In Eastern countries the record is black. Egypt still heads the list, mainly because of trachoma. Tunis and Algeria come next, again with a high incidence of trachoma. India and China follow with many cases of malnutrition as well as trachoma. Many of the nutritional cases are due to deficiency of Vitamin A. Among the so-called civilised races, New Zealand shows the best record with only 0.3 per thousand, followed by Switzerland, Holland and Germany with 0.5 (1937) and U.S.A. and Great Britain with one per thousand of the population.

The whole point of giving these inaccurate figures (there are no accurate statistics on these points) is to draw a parallel between economic conditions or standards of living and incidence of blindness. It is quite obvious that in Egypt, India and China, where shortage

of essential foodstuffs among the general population is acute and living conditions, to a European, are impossible, no amount of medical care or provision of hospitals can deal with the situation. Only if doctors could write prescriptions for food and the hospital dispensaries could ensure that it was eaten by the patient would the situation improve. Vitamin tablets even will not replace good food. Indeed, one might almost say that the incidence of blindness in the population of a country is a good indication of the standard of living of the people. There is, of course, a certain minimum of blindness which cannot be reduced (due to congenital defects and unavoidable accidents), but short of that we can say that infections and nutritional diseases and industrial accidents can all be considerably reduced by raising the standard of living and by education. This will take us a very long time judging by the present state of the world.

Let us now consider what blind persons can do and how they can be taught. In countries such as Egypt and the East in general, where eye disease is much more common than it is here, the blind have from time immemorial been able to take their place in society as musicians, story-tellers and masseurs. Their capabilities are understood and people are used to them. In England, however, with its fifty million inhabitants and only about 770,000 blind persons (or Australia with only about 4,000 to 5,000 blind) it may be that many people have never met and known anyone who is blind. A blind person therefore is likely to be regarded by them with excessive pity, admiration or fear. Pity is probably universal, since everyone feels how awful it would be to become blind. Most people, if asked, would say that it is far worse to become blind than stone-

deaf, but in the experience of doctors it is the deaf person who is the far more unhappy, because he is more cut off from human intercourse and is treated much more unkindly by society as a whole. The blind person arouses sympathy and so is sure of help, often clumsy and misplaced, it is true, but almost always willingly given. He is not cut off from social pleasures and is capable of a relatively independent life.

In some people the blind arouse great admiration. Their fascinating ability to read with their fingers and their usual cheerfulness lead often to quite foolishly exaggerated accounts of their capabilities. Their sense of touch is said to be abnormally acute, their memories phenomenal beyond belief, and their possession of some completely mysterious sixth sense asserted by their admirers. In some people, however, they arouse a feeling of revulsion. Their often curious looking eyes and slightly expressionless faces, which respond to the tone of the speaker's voice and not to his facial play, which may be at variance with this, are slightly eerie. Some people, going to the other extreme, feel them to be essentially stupid, and blind children are often treated as if they were also mentally deficient. It often seems very difficult for the ordinary mortal to steer a middle course between adulation and insult. All that is required is actually a little imagination and commonsense. Each blind person is an individual with a basic character of his own, grave or gay, intellectual or stupid, honest or otherwise, placid or irritable, and so on. Most people fail to recognise that this will not be altered by his blindness. The whole individuality is so overshadowed by the fact that the man is blind that they cannot see what kind of a person he really is. Try talking to a blind person with your own eyes shut.

This will often make it easier to sense his natural character.

It is, of course, on this basic character that the blind man's response to his blindness depends. You cannot teach Braille to a person who has never had any desire to read, nor can you expect the same behaviour from a healthy vital youngster born blind as from a businessman blinded by glaucoma in middle life. This is self-evident yet often forgotten.

Let us consider the chances the blind child has of being educated and of achieving an independent life. In the year 1946 in England there were 909 blind children of school age being educated in schools for the blind, 37 in other schools and 105 not in any school. Some of the very young children are with their own families but a number are in special nursery homes (Sunshine Homes) run by the National Institute for the Blind. In England and Wales there are 27 elementary schools for the blind, three secondary schools, one school for retarded blind children and one for blind mental defectives. It is thought in England that unless the parents of a blind baby are of superior intelligence they are apt to retard its development by over-protecting it, and it is usually better for them to send it to a home. There are, however, only one or two homes for pre-school blind children in Australia and so they are usually kept with their parents until they are five.

When the child reaches school age there are various methods of education open, depending on the child's intelligence. There are in England about 40 schools for the education of blind and partially sighted children. Some are run by the National Institute for the Blind, others are under the County and Borough Councils and others are incorporated charities. In Australia

the procedure varies from State to State in that some of the schools are privately endowed and others are State schools under the Department of Education. In some there will be found a class for blind children attached to an ordinary State school, in others there may be a boarding school for blind children only. There are eight schools for the blind in Australia and all the States except the Northern Territory are provided for. In most of the schools primary education only is aimed at. Intelligent pupils who wish to go further can attend sighted secondary and modern schools and universities. There are no special schools for those children who are mentally retarded. The numbers of these children are small and the distances in Australia so great that the English system cannot at present be fully implemented. In some of the States, however, there are classes for partially sighted children who see just too well to be taught Braille but not well enough to compete with fully sighted children in a large class. These classes are usually very useful in helping the child to overcome his handicap and to take a more or less normal place in the sighted world. Since the classes are small, the tuition is much more individual than in the normal classes and the children's attainments are often very good. In England and Australia it therefore usually comes about that the education received by a blind child depends more on its intelligence than on its social status. Since there are relatively few schools to choose from, it follows that all the blind children of one area or State gravitate to one school. During their elementary school years they sort themselves out according to their intelligence and some may pass on to secondary or high schools and even to the universities. In Australia (as in parts of the United

States also) it is often the practice to send a clever blind child to a sighted school once he has learned Braille and touch typing. This helps much in his social integration and gives him a chance to develop to his full capacity.

In Australia, blind students have amply proved themselves and a truly surprising variety of occupations are now open to them. The following list is probably not exhaustive but will give some idea of the versatility of the blind:

University lecturer.
Barrister and solicitor.
Minister of religion.
Teacher.
Welfare worker.
Speech therapist.
Physiotherapist.
Accountant.
Musician.
Radio compere.
Breeder and judge of cattle.
Orchardist.
Poultry farmer.
Newspaper reporter.
Telephone operator.
Shorthand-typist.
Carpenter and cabinet maker.
Gardener.
Dark-room worker.
Process worker in industry.
Salesman.
Brush, mat and basket worker.

The number of blind persons graduating from universities of Great Britain has been steadily rising

but is naturally small since these students represent the pick of blind children, of whom there are relatively few. Between 1905 and first World War, 32 students graduated, between 1915 and 1924 there were 31 and from 1925 to 1934 there were 50. Such professions as the law, teaching, music and massage, figure in their subsequent careers, as also in Australia. The majority in England took their degrees at Oxford, the remainder at Cambridge, Durham, Edinburgh and London. That the degree of scholarship aimed at is high is shown by the fact that 69 per cent. of the blind students at Oxford read for honours. Seventeen per-cent. of them took a first class as against eight percent. of the sighted students. This is to be expected as they represent a highly selected group. This careful selection is essential both to avoid discouragement and disappointment to the blind students themselves, and also to ensure that no failure occurs to detract from their reputation as a class, since if a blind student fails it is likely to be put down to his blindness and not to his stupidity or laziness. It will then act to the detriment of all the other blind. The standard aimed at and attained has therefore to be high in order to convince the sighted world that higher education is possible for the people who are unable to read ordinary books.

How are blind children taught? The answer naturally is orally and by touch. There have been various systems of embossed type invented in the past, of which only two—Braille and Moon—have stood the test of experience. Louis Braille was a blind Frenchman about whom little is known, save that in 1834 he invented a system of writing by embossing raised dots on thick paper, which has since become the universal medium of education of the blind. The system of writing is

logical and easy. Each letter consists of from one to six raised dots in two parallel lines of three, like this ⠇ These are numbered 1 4 / 2 5 / 3 6. The possible combinations of these six dots give 63 different signs. The first 10 signs are the letters A to J, and use dots only in spaces 1, 2, 4 and 5. Thus A is 1, B is 1-2, C is 1-4, etc. The second ten signs are the letters K to T and are formed by adding dot 3 to each of the signs on the top line. Letters U, V, X, Y and Z are then made up adding dots 3 and 6 to the signs in the top row. As Louis Braille was a Frenchman, the sign for W is an exception as it does not occur in French and has to be added when writing English. It is made of dots 2, 4, 5 and 6. The rest of the 63 signs are used for punctuation marks, for fraction lines, numeral signs, decimal points and for contractions of commonly recurring words and combinations of letters, such as ch, gh, sh, th, wh, ed, er, and, for, of, the, with, etc.; by further contracting and abbreviating it is possible to save more space and write more quickly as skill is attained. A highly contracted form (Braille shorthand) exists which can be written by an expert at the rate of 130 words a minute.

The writing is done with a small stylet on a special board having a series of perforations on a metal band which keeps the writing straight. As the dots are made by pressing the stylet on to the paper, the raised letters formed will be read by feeling the reverse side of the paper. It follows, therefore, that the letters must be reversed and written from right to left. For example, CAT is written thus ⠉⠁⠞ but felt like this ⠞⠁⠉ when read. A sort of typewriter having six keys cor-

responding to the six Braille dots is used also and for shorthand a small mechanical writer taking a narrow strip of paper, like ticker tape rolled up. This makes for speed, as no time is lost in changing the paper or sliding the metal band down the page.

During the first World War a most ingenious appliance was invented by Dr. Fournier d'Albe. It was called an optophone and its object was to enable a blind person to read an ordinary printed book. The principle involved was the transformation of light into sound. Essentially the apparatus consisted of a lens system which focused a picture of the printed letters one by one on to a row of minute photo-electric cells arranged in a vertical row. As each letter cast its shadow on the cells the amount of current passing through the cells was altered by the alteration of illumination due to the black and white parts of the letters, and the changing pattern caused by the successive letters passing over the electric "eye" was transformed into a series of musical notes. Each letter had, as it were, its own sound pattern, which had to be learned and could then be interpreted back into letters and words by the trained operator. A good musical ear was essential and the technique was difficult to master, only a few people becoming proficient enough to read with any fluency by this means. Owing to cost of manufacture and difficulty of manipulation the instrument has fallen into disuse, but it is a most ingenious invention and with modern advances in physics may yet be resuscitated and simplified. It was occasionally used at blind schools with but little success, about twelve people having been taught to read with it, and one, Miss Jameson, having attained a proficiency of 100 words a minute. She did much to further its more extended

use and received the O.B.E. for her services to the blind in this connection. Its chief disadvantage is that reading is letter by letter, and one cannot tell what is coming: both with sighted reading and Braille the word ahead is being recognised in advance all the time.

Children in all blind schools are taught to use an ordinary typewriter for writing to their sighted parents and friends, and for using in examinations, etc. The use of a typewriter is easy, since all good sighted typists work by touch alone and do not need to look at the letters on the keys.

Arithmetic in blind schools is done on a special board perforated with eight-sided holes into which fit small metal pegs with embossed ends. Each peg can occupy sixteen different positions from which all the numerals and signs can be obtained. Algebra is done in a similar way, using only eight signs.

Geometry is always rather a difficult subject for the blind. In some schools the diagrams are made in fine wire stretched on pins on a sort of cushion. In others they are pricked on Braille paper with a "spur wheel" with which lines and circles of embossed dots can be drawn. It is also possible, by writing with a stylet on cellophane stretched on a rubber pad, to produce lines which can be felt from the right side of the paper, not the underneath as in Braille writing on a board. This principal is much used in the United States. It is distributed by the American Foundation for the Blind and is called the Sewell Raised Line Drawing Kit. It has an even greater value, however, in that blind children can be taught to write in ordinary handwriting with it and can then use a ball-pointed pen to write on ordinary paper and can (which is even more useful) learn to sign their names.

Geography is taught by means of embossed maps and globes of various kinds.

Music is always a popular subject. The Royal Normal College for the Blind in England specialises in the teaching of music, and some years ago organised a school orchestra which toured England and the Continent with great success.

Languages, history and elementary science, including botany, zoology, chemistry and physics are also taught in the secondary schools and all the schools teach handicrafts.

Physical exercises are an important item in the education of the blind: their physical development is often poor and blind children tend to develop many undesirable tricks of posture and habit which require correction. Drill, dancing and games all have their place in the curriculum. The teachers in these classes must obviously be sighted as, indeed, must most of the staff in blind schools if they are to be run efficiently and the children are to be thoroughly looked after. A few teachers in each school are usually blind, or partially sighted. Of 145 blind or partially sighted men and women graduating from universities during the fifty years 1884–1934, twenty-four adopted teaching in some form or other as their profession.

Those children whose progress at school does not justify their being sent to a university are eligible for a variety of occupations ranging from manual work, such as brush, rug and basket-making, machine knitting, gardening, poultry farming and certain factory processes to the rather less manual work of telephone switchboard operator and shorthand typist. Massage also forms an extremely suitable type of work for the blind. In Australia, owing to the small number of

blind children, the facilities for higher education are not particularly good. It is easier for an intending masseur to go to England for training than to try to obtain it in a sighted physio-therapy school in Australia. In London, for example, there is a school for blind students only and a large clinic (the Eicholtz Clinic) staffed entirely by them.

Since the beginning of the Second World War the number of possible occupations for the blind has been increased in England and Australia mainly by the efforts of the National Institute for the Blind, which has successfully placed them in various industries where they had never proved their worth as workers before. During the war over a thousand registered blind were placed in various war factories. They worked on such processes as burr filing, working to set stops and jigs, set gauge and aural inspection operations, repetition assembly and packaging and the smoothing of rough surfaces in metal, wood or plastics. An investigation by the National Institute has culminated, in England, in a national scheme for their employment which is operated through the labour exchanges with the help of the local agencies for the blind.

Certain names of blind men and women stand out in the history of the improvement of the education and opportunities for the blind. Many systems of embossed writing have been invented (Frere, Alston and others), but only two have stood the test of time—Braille, in which there exists a library of thousands of volumes (about 800 books a year are added to it) and Moon, which is much easier to learn and therefore popular among the elderly blind. This system was invented by William Moon, who was born 120 years ago, and who

started a small school for blind children in Brighton. His experience of the difficulty of teaching the Frere type (which is phonetic) to children led him to invent a simpler system which had immediate success. Between four and five thousand books are available in this type.

It is to Dr. Armitage and Sir Francis Campbell, both partially sighted, that the blind owe many of the present facilities for education. The former as the founder and the latter as the first head of the Royal Normal College for the Blind in England, realised to the full the importance of a liberal education, including physical training and music. The school they founded has become world famous, and is still the principal centre for musical study and also for the training of piano tuners, as this was one of the earliest paid jobs available to blind people.

To Elizabeth Gilbert, blind daughter of a Bishop of Chichester, is due the credit of establishing the first workshop for the blind and the first home workers' scheme. This was in 1854 and the following year she founded the Association for Promoting the General Welfare of the Blind, which afterwards grew into a prosperous trading concern. Its shops are seen in all the big towns of England today. Similar organisations also now exist in all the capital cities of Australia.

In more recent years the names of Sir Arthur Pearson and Sir Ian Fraser stand out in connection with the National Institute for the Blind and St. Dunstan's in London. Early in 1914 Pearson became Treasurer of the Institute and raised £30,000 to rebuild it and to form a fund for producing cheap books in embossed type. During the First World War he opened a hostel for blinded service men which after-

wards grew into the organisation known as St. Dunstan's, where he was succeeded by Sir (then Captain) Ian Fraser, who had himself been blinded in the war and trained by St. Dunstan's. This organisation also rendered valuable service in World War II and in peacetime continues to help in the rehabilitation of newly blinded civilians.

In addition to these considerations about people already blind we should think a little about the means existing for dealing with eye defects and diseases, which are not blinding but which must be treated.

First of all let us see how knowledge about the eye is to be got. Take the study of ophthalmology (or everything to do with eyes). Who studies it and where and how? We will begin with the doctors. Their affairs are just as complicated as those of everyone else. The would-be doctor passes a preliminary examination in general subjects to show that he has enough brains to be allowed to begin and then enters a medical school. Here he studies approximately six years, learning first through chemistry, physics and biology, a little of how the world of sense is put together, and then through anatomy and physiology of how the human body is related to the rest of this world, as we know it, what its structure is and how it works. He then studies pathology, which tells him of the principles of the disease processes which may afflict us, therapeutics or how to cure some of them and finally he studies diseases proper, of mind and body, by observing actual patients in hospital. He passes various examinations on the way and finally emerges with either a University degree in Medicine and Surgery, or a diploma from one of the licensing bodies in England (i.e. the Royal College of Physicians, the Royal Col-

lege of Surgeons or the Society of Apothecaries). In Australia the qualification is a degree from one of the State universities. This entitles him to have his name placed on the register kept by the General Medical Council in London, which entitles him to a certain legal position. He can sign death certificates and can prescribe 'dangerous drugs': in return he is entitled to legal protection.

He is now an ordinary doctor, and knows a very little about the eye in a sketchy sort of way. If he wishes to become an eye specialist he must study another two or three years and take some more examinations. He is now an ophthalmologist or ophthalmic surgeon or oculist and can diagnose and treat eye diseases, can operate on the eye, can test for spectacles and has, in fact, a grasp of the whole subject.

In England there are special hospitals for eye diseases only, as well as eye departments in all the large general hospitals. In Australia all the teaching hospitals include eye departments but there are no special eye hospitals, though the Victorian Eye and Ear Hospital in Melbourne serves much the same purpose. It treats ear disease as well. There are also eye departments in many non-teaching hospitals outside the capital cities especially in Victoria and Queensland.

The ophthalmologists often have working with them Medical Auxiliaries called orthoptists. Medical Auxiliaries are people who, without being doctors, have been trained in certain forms of medical treatment and diagnosis, such as massage and X-ray photography. They have a register in England under the British Medical Association and work with doctors either in hospitals or in private practice. Orthoptists are trained in giving treatment by exercises for the develop-

ment of and strengthening of binocular vision (see Chapter XII). They have been trained for two years in an eye hospital or in an orthoptic school attached to an eye department in a general hospital. In addition to working with the ophthalmologists they may teach in an orthoptic school. These orthoptists are of course quite apart from the various systems of eye exercises which come into vogue from time to time. Orthoptists are never allowed to give exercises without the supervision of a doctor.

In addition to the ophthalmologists there is another essential link in the chain between the patient and his cure, namely, the optician. Opticians are of two sorts, optometrists and dispensing opticians. The latter, who are rarer in Australia than in England, where they are well organised into a Guild, manufacture lenses and spectacle frames. They have shops to which the patient can take the prescription given him for glasses by the ophthalmologist (oculist).

They are responsible for the accurate grinding of the lenses to the prescription and for their accurate fitting to the patient's face. No matter how accurate the doctor's prescription, the glasses will not give complete relief unless they are fitted so that they are the right distance from the eyes and properly centred. Dispensing opticians may also make optical instruments used by doctors (e.g. ophthalmoscopes, magnifying glasses and so on).

Optometrists or sight testing opticians as they are called in England, on the other hand, pass a different kind of examination and undergo training in how to order the correcting spectacles for any error of refraction. They learn to examine the inside of the eye and to recognise anything unusual. As however, they have

had no medical training, they find it difficult to interpret what they see and to link it up with the patient's general condition. They are also unlikely to be able to estimate the amount of connection, if any, between the patient's error of refraction and his symptoms.

Let us now consider the reasons which lead the man in the street to seek advice about his eyes. The deciding factor is usually one of six things.

First, some people go themselves and take their children for a check-up in the same way as one goes to a dentist, in order to 'take things in time'. This is quite a good plan provided it is not indulged in too often, since healthy eyes can usually be trusted to get on much longer without attention than can teeth. Children can with advantage be examined twice between the ages of four and fourteen, unless some obvious defect such as a discharging eye or a squint is present, when immediate attention is necessary. A good oculist can often foretell by the age of twenty or so at what period of life the next test should become necessary and glasses (except in growing children) hardly ever need changing more than once in three years and frequently much less often than this.

Secondly, an injury will require immediate attention. Many people if they get a bit of grit in the eye and fail to remove it by the usual means (finger, mirror and handkerchief, assistance of friend, etc.) go first to a chemist's shop. No one quite knows why this is, since chemists receive no special training in removing things from eyes. Other people go more wisely to their family doctor, who can usually help them but who may not have the necessary lights and magnifying glasses to find and remove a very small foreign body. The thing to do if you are in a city is to go

straight to the hospital (the eye hospital if there is one or the eye department of the general hospital) where there is usually a house surgeon who is expert in getting out bits of grit. In some of the big hospitals in industrial towns the house surgeons may remove more than a hundred bits of grit in a day. Everyone realises the urgency of the case and tries to deal with it promptly. This is well brought out in an annotation in the Lancet in 1939 apropos of closing the eye hospitals in Central London in case of air raids. The writer pointed out that a piece of grit in the eye was a 'surgical emergency' and wrote 'a foreign body under the upper lid will take your mind off any war'. This applies to other injuries of the eyes as well.

Thirdly, a parent may have noticed something wrong with his or her children's eyes, a stye, a red eye, a discharge, a squint or something like that. These are obviously not jobs which can be dealt with by a chemist or an optometrist.

Fourthly, the patient may be having headaches, although his sight is perfect and his doctor may suggest that his eyes should be tested. As explained already, it is advisable for this to be done by the oculist himself in case the cause is not solely an error of refraction, though this may be present as well. In many places in Australia, however, it is not easy for the patient to get to an eye doctor so the general practitioner must try to assess the optometrist's findings against the patient's symptoms. Obviously if a headache disappears after one starts wearing glasses it is practically certain that the need for them was its cause.

Fifthly, the patient may notice that he does not see as well as he could wish. He is very apt to go straight to an optometrist and buy a pair of glasses. The

reasons for and against this procedure must be decided on the merits of the case.

Sixthly, the victim may notice that he has some disturbance of vision, he sees black spots, jazzy lights or objects look bent or distorted, or there are bits missing from the field of vision. There can be no doubt that this requires careful examination and diagnosis by an eye doctor.

In most cases the skill of the ophthalmologist and the optician between them will be able to cure or relieve the condition completely. For a few unfortunate people, however, nothing can be done and it is then necessary for us do do all in our power to render their lives as full and independent as possible in spite of their handicap. In many of these cases greater scientific knowledge or earlier treatment might have prevented their blindness, and it is to the field of prevention that we should devote our energies. In the past we have spent practically nothing on planned prevention of blindness, either by education of the public in the basic facts about eyes, which would enable them to seek advice in the early stages of the disease or by improving facilities for research, which should lead to greater knowledge and to the possibility of prevention or cure of many of the present still mysterious diseases. In England and Australia since the last war institutes have come into being whose sole function is research into the nature and causes of eye disease. One of these is a part of the University of London and works in close association with the large London eye hospitals. There are others in other University towns (Oxford, Birmingham etc.). In Australia the movement is also on foot as is shown by the establishment of the Ophthalmic Research Institute of Australia with

its headquarters in Melbourne and subsidised research workers in other cities. Interest in the subject has grown tremendously since advances in treatment have been possible through such things as the discovery of the anti-biotics and cortisone. Now, whenever a discovery is made in any branch of science, ophthalmic research workers investigate it to see if it has any application to eye disease.

We have tried in this book to set out the fascinating complexity of the eye and the mechanism of vision. The gaps in our knowledge both of the healthy and the diseased eye are painfully obvious, for example, what are the causes of cataract, how can one prevent corneal scarring, what caused the loss of sight in Japanese prison camps? Such problems still present themselves and can be solved given time, if enthusiastic research workers, financial help and facilities for investigation are available. Today even in England and the United States where there are so many competing claims for money, it will take time for reorganisation of hospital services, training of eye specialists, of basic scientific workers and provision of research laboratories. It is significant that in the past we have spent practically nothing, especially in Australia, on planned prevention of blindness, either by improving the facilities for treatment, by educating the public in the basic facts about eyes or by research.

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